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XXVI

ALIPHATIC VERSUS AROMATIC AMINES AS VASOCONSTRICTORS

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The treatment of any disease in its best form is directed to the cause. This principle unquestionably also holds true with the affections of the nose and accessory sinuses characterized by turgescence of mucous membrane. Unfortunately etiological treatment of these conditions is either unyielding or not available. We, therefore, have to resort to palliative measures. According to Proetz,[1] Jelinek used cocaine for the first time on the nose in 1884, the same year Koller[2] discovered its local anesthetic action in ophthalmology. Proetz also mentioned that Velich and Königstein first applied an epinephrine solution to the nose in 1897, the same year Abel announced his attempts to isolate epinephrine in pure form.[3] For over a quarter of a century, these two products, cocaine and epinephrine, were the only vasoconstrictors to give temporary relief to sufferers of rhinitis. The comfort to the patient, facilitation of nasal drainage and reduction of anoxemia as a result of such palliative therapy cannot be underestimated. It was gradually recognized that cocaine produces addiction upon repeated use and that occasionally the amount of cocaine absorbed from the nasal mucosa causes toxic manifestations and death. By injection epinephrine is a perfectly useful drug in the

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management of allergic diseases and in combination with local anesthetics for surgical purposes, but upon direct application to the nose it is too short-acting and induces secondary congestion which is often worse than before treatment.

It was thus not surprising that after Alice H. Cook, Fetterolf, and Sponsler[4] demonstrated the vasoconstrictor action of ephedrine on the human nose, cocaine and epinephrine were promptly abandoned. Their results indicated that the action of ephedrine when applied locally to the nose lasted for about 3¼ hours—with no signs of irritation. Equally convincing are the animal data. Fig. 1 shows the blood pressure tracing of a pithed dog following an intravenous injection of ephedrine. During the rise of blood pres-

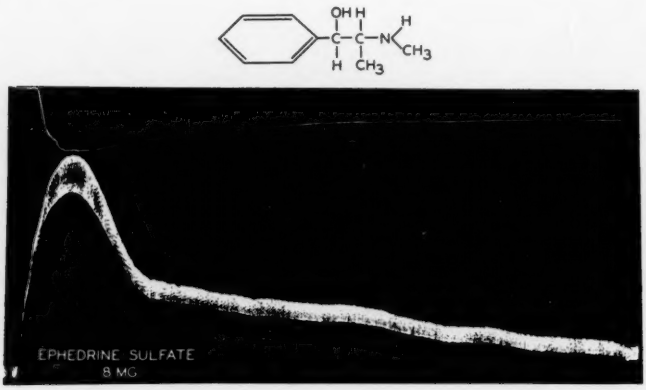


Fig. 1.—Action of ephedrine on the blood pressure and nasal volume of a pithed dog.

sure, the nasal volume diminished, indicating the contraction of mucous membrane of the nose and accessory sinuses—recorded by the Jackson technique.[5] Complete return of blood pressure in this experiment occurred in 57 minutes. In human subjects, a dose of 60 mg. given by mouth caused circulatory changes for more than two hours.[6] This long duration of action, as well as effectiveness by mouth, is due to the fact that the enzyme amine oxidase, which is abundantly present in the body, does not destroy ephedrine.[7] The drug is slowly excreted in the urine. Evidence is accumulating to show that the methyl group attached to the alpha carbon atom, from the amino group, protects the compound from being attacked by amine oxidase.[8, 9]

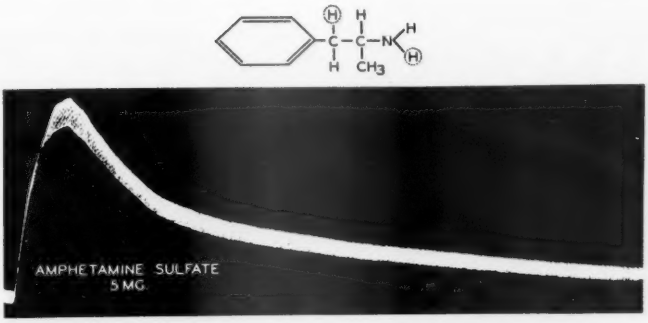
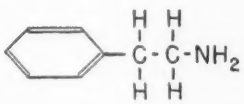


Fig. 2.—Action of amphetamine on the blood pressure of a pithed dog.

Ephedrine is a relatively simple substance in the opinion of organic chemists. It has a benzene ring and a side chain of three C-atoms, with a methylamino group attached to the middle C-atom (Fig. 1). It therefore belongs to the aromatic amine series. Among the older aromatic amines which can be readily prepared synthetically is β-phenylethylamine. Investigations in animals soon revealed that β-phenylethylamine has a short action, and is not effective by mouth.[10] Clinical work by Fenton and Larsell[11] fully substantiates the laboratory data, and rules it out as a suitable drug. By looking at its structural formula, we can understand the reason why:



β-Phenylethylamine has a side chain of two C-atoms; there is no methyl group on the α-C-atom to inhibit the action of amine oxidase.[12-14] It is thus, in contrast with ephedrine, quickly eliminated in the body.

Newer aromatic amines were then provided by chemists. Of the numerous variants, propadrine has an action most comparable

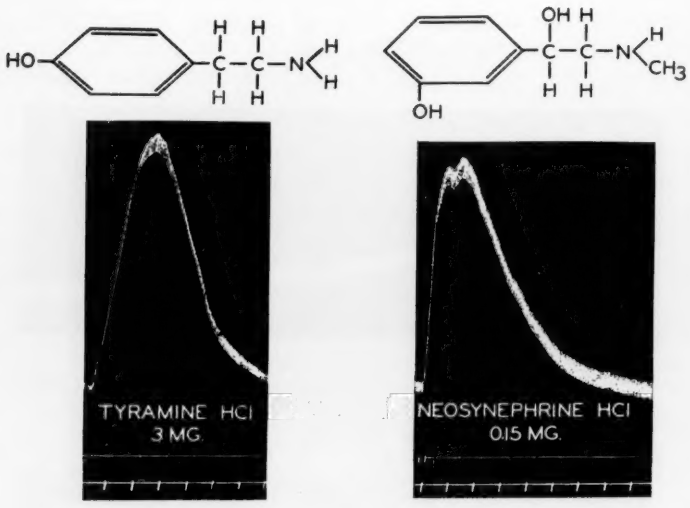
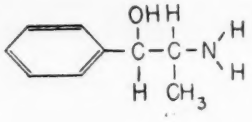


Fig. 3.—Action of tyramine and neo-synephrine on the blood pressure of a pithed dog.

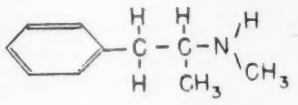
to ephedrine. This is understandable, because it differs from ephedrine by only one methyl group on the amino-N, as shown below:



Another widely-known product is amphetamine or benzedrine. In Fig. 2, it can be noted that it has a long duration of action and it is effective by mouth, because of the protective action from amine oxidase of the methyl group on the α-C-atom. Unfortunately amphetamine has a dominantly stimulating action on the central nervous system as compared with its vasoconstricting action, which owes its origin to the stimulation of the sympathetic nervous system. Apparently the replacement of the hydroxy group on the β-C-atom by a H-atom results in a compound having a special affinity for the brain and spinal cord (Fig. 2). During World War II the stimulating action of amphetamine on the central nervous system certainly justified its purpose, for General Grant[15] of the American Air Force

said, "Benzedrine is the most satisfactory drug now available for temporarily postponing sleep when the desire for sleep endangers the security of a mission." On the other hand, amphetamine is becoming less and less desirable in otolaryngology in view of the central stimulation, addiction liabilities,[16] and relatively high toxicity.[17-21]

The Germans during World War II used pervitin, known in this country as desoxyephedrine, for alerting purposes in their air corps. If the following formula is examined, the close similarity of its structure to that of amphetamine is obvious:



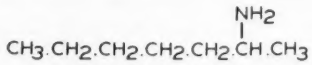
The disappearance of the hydroxy group on the β-C-atom again confers a marked degree of central stimulation.

In search for newer vasoconstrictors the analogues of epinephrine were not forgotten. The two phenolic hydroxy groups add instability and bring about rapid destruction by phenolase and other enzymatic oxidation systems.[22-24] Even a single phenolic hydroxy group, as in the case of tyramine and neo-synephrine (Fig. 3), cuts short the vasoconstricting action and nullifies the systemic effects after oral administration. Paredrine belongs to the same class. It thus appears clear that in spite of the numerous aromatic amines synthesized, there are very few which can be truly called good vasoconstrictors.

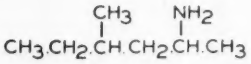
Chemical studies were extended to other fields by synthesis.[25, 26] It was soon observed that the benzene ring is unnecessary; in fact amines of the straight chain series have the same type of vasoconstriction. These are known chemically as aliphatic amines. Here again, a methyl group on the α-C-atom confers a prolonged action since it protects the compound from oxidation by amine oxidase.[27] One of the potent compounds, tuamine, has a length of seven C-atoms. Its clinical possibilities were explored by Proetz.[1, 28] If the methyl group at the end is transferred to C_4, the resulting compound, fouramine, is 13 per cent more active than tuamine. Neither product is effective by mouth, nor stimulates the central nervous system in therapeutic doses.

Compounds of shorter or longer chains are invariably less active. Thus, one of eight C-atoms has only 1/6 the activity of tuamine.

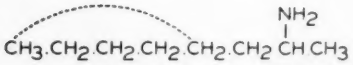
For sake of clearness the following formulas are listed:



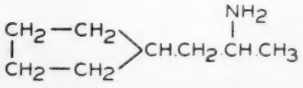
TUAMINE



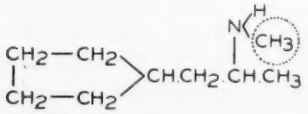
FOURAMINE



1-PENTYL-2-AMINO-PROPANE



1-CYCLOPENTYL-2-AMINO-PROPANE



1-CYCLOPENTYL-2-METHYLAMINO-PROPANE (NO 02040)

If the terminal C-atom joins the fifth C-atom to form a ring, as indicated, the product approaches fouramine in activity as measured by the blood pressure response. Further, if a methyl group is introduced to the amino group, the resulting substance, 1-cyclopentyl-2-methylamino-propane, becomes 56 per cent more active than fouramine. Judged by peak of action, it is more potent than ephedrine. As yet no simple name has been assigned to the compound, and for brevity it is known to us by its serial number 02040.

A sample experiment on blood pressure is shown in Fig. 4. No. 02040 has the same duration of action as ephedrine. When given by mouth, it is more easily absorbed than ephedrine.[29] A rise of blood pressure is detectable in human subjects with an oral dose of 50 mg. of the hydrochloride. It does not cause immediate inhibition of the ciliary movements of the rabbit's isolated trachea, as studied by the Proetz technique.[30, 31] Table 1 shows its comparison with ephedrine, tuamine, and saline control.

Compound No. 02040 has a slight stimulating action on the central nervous system. The activity curves of rats indicate that it is 1/40 as active as amphetamine recorded by the modification[32] of the spring-suspended cage technique originally designed by Schulte and associates.[33] Ephedrine studied under the same conditions is 1/10 as active as amphetamine on the central nervous system. It appears from our laboratory data that No. 02040 is better all-around than other vasoconstrictors of either the aromatic or alipathic series.

Compound No. 02040 dilates the rabbit's pupil when instilled into the eye. This is to be expected, because any sympathomimetic

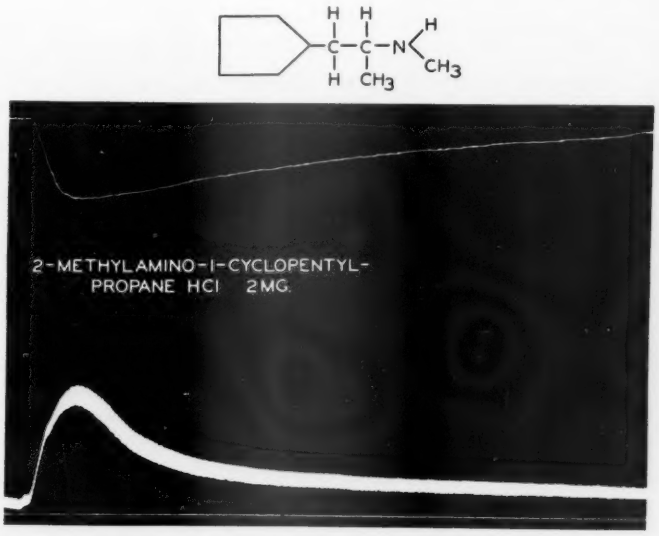


Fig. 4.—Action of 1-cyclopentyl-2-methylamino-propane (No. 02040) on the blood pressure and nasal volume of a pithed dog.

amine stimulates the endings of radial dilator fibers. Mydriasis also occurs in man if a 5% solution of the hydrochloride is dropped into the eye, with no cycloplegia. There is a racial difference of response, however; the pupil of the Caucasian dilates, that of the negro is refractory, and that of the Chinese reacts slightly—exactly similar to cocaine or ephedrine, as far as the selective action on the human eye is concerned.[34]

For over 20 years, vasoconstrictors have been directly applied to the human nose in a great variety of pharmaceutical forms. There

TABLE 1.—ACTION OF VASOCONSTRICTORS ON THE CILIARY ACTIVITY OF THE ISOLATED RABBIT'S TRACHEA.

Drug	Concentration	Number of Specimens Studied	Mean# Duration of Ciliary Activity	
	percent		hours	minutes
Ephedrine Sulfate	0.5	6	5	38
	1.0	6	3	53
	2.0	6	2	53
Tuamine Sulfate	0.5	6	3	19
	1.0	6	3	54
	2.0	6	1	19
1-Cyclopentyl-2-methylamino-propane Hydrochloride	0.5	6	3	43
	1.0	6	2	36
	2.0	6	1	24
Sodium Chloride	0.9	12	17	48

#This is the geometrical mean. According to statistical principles, the geometrical mean, which is the antilogarithm of the average of logarithms of individual readings, is more reliable than the arithmetic mean.

is a trend that they are used altogether too frequently. Certain patients actually spray their own noses every few minutes in spite of the physician's warning. This sort of self-medication defeats the very purpose of modern therapy. In the first place, all the long-acting vasoconstrictors show tachyphylaxis, namely, they produce very little response if they are repeated before the effects of a previous dose wear off. Secondly, incessant administration of these nose preparations results in absorption of the active ingredients with the occurrence of side reactions due to central stimulation, such as nervousness, a "jittery" feeling, and insomnia. Thirdly, too frequent medication with vasoconstrictors may constitute a factor predisposing to middle ear complications. Such dangers have been emphasized by Kully.[35]

In 1926-27, Gaarde and Maytum[36, 37] treated ragweed hay fever patients with ephedrine by mouth and they observed a decrease of nasal secretion. They also stated that ephedrine as a nasal spray was less efficacious, and the relief was of shorter duration. Since their publications I have personally confirmed their results from season to season—namely, by oral use of the drug only. In other words, vasoconstriction of the nose can be accomplished by the systemic effect of ephedrine. This may apply to other drugs, including No. 02040. I submit this proposition because I believe that if these drugs are prescribed by mouth twice or three times a day, better results may be forthcoming in otolaryngology.

During the last two years, there has been increasing evidence that allergic rhinitis responds favorably to antihistaminic drugs. The question arises as to which type of drug is the more effective—vasoconstrictors or antihistaminics. The answer will not be available until some carefully controlled comparisons have been made. From a pharmacologic point of view, there may be a synergism between the two—one by sympathetic stimulation, and the other by neutralization of histamine, a product of the allergic reaction. It is not impossible to visualize that simultaneous administration of both types of drugs, by mouth, will give rise to better results in such conditions, than with either alone. The correction of wakefulness, which is inherent in some vasoconstrictors, by the sedative action of antihistaminics, would favor such a combination. A preliminary report[38] seems to substantiate this contention.

The author is deeply indebted to Mr. E. E. Swanson, Dr. Francis G. Henderson, and Dr. Paul N. Harris for assistance in the preparation of this manuscript. Thanks are also due to Miss Marian H. Ellaby and Mr. Harold M. Worth for the preparation of the illustrations.

LILLY RESEARCH LABORATORIES.

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XXVII

NEURON ARCS OF CLINICAL SIGNIFICANCE IN LARYNGOLOGY

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An uncommon manifestation of interest can hardly be expected to well up among clinicians over a complex anatomical subject of this character, yet it is hoped that it will serve in a measure as a basis for accurate thinking in some of the practical aspects of laryngology. Specifically the text treats of certain respiratory reflexes and the neuron pathways along which they travel. An attempt is made to describe various nerve circuits composed of peripheral, nuclear, and association routes and to explain by reason of their position, relationships and function, some of the phenomena observed in clinical practice.

While much of this subject may be only of academic interest, it is nevertheless axiomatic that the basic sciences lay the foundation of medicine. From a knowledge of them one may travel in any direction. A veritable panorama of opportunity is exposed to the individual who is grounded in the fundamental principles of some phase of science: the opportunity for intelligent observation, research, the development of therapeutic facts, and the ability to put these facts into discriminate practical usage. It may not be too much to expect, therefore, that this anatomical study of some of the reflexes of respiration may serve as a common core about which a better understanding of respiratory symptomatology and a greater knowledge of rational methods of treatment may be developed.

Of the literature on respiration only source material for the figures is quoted. The pontine pneumotaxic center[1] (which may be slightly farther forward than indicated in Fig. 1, although its position in man is not known) has been described repeatedly. The

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Presented before the meeting of the American Laryngological Association, Hot Springs, Va., April 14, 1948.

diagrams show, in the medullar respiratory center,[1] only the bordering zone of the expiratory field which coexists with and surrounds the inspiratory field. Pitts, Magoun and Ranson[2] discussed the rythmic character of respiration as dependent upon the similar and "mutually replaceable" activity of (1) the vagal arc stimulated by lung stretch, and (2) the pneumotaxic center. Gesell[3] stressed the influence of stretch impulses from skeletal muscle. The descending pathways from cortex[4-6] and hypothalamus[7] (Fig. 1) to the brain stem respiratory centers are a part of the generally recognized anatomic mechanism for emotional expression.[8]

1. *The Neuron Paths from Cortex to Cord Concerned with Increasing Respiration* (*Fig.* 1). The frontal cortex is connected with the preoptic area and rostral part of the hypothalamus, either (1) directly by corticohypothalamic fibers (Mettler[6]) or (2) by two neuron arcs (corticothalamic and thalamohypothalamic tracts) with synapses in the dorsal thalamus.

A. From the caudoventral preoptic and anterior hypothalamic transition area (and from the hypothalamic area farther caudalward in the region of the dorsomedial hypothalamic nucleus, in our own material) fibers are contributed to the dorsal longitudinal fasciculus. These fibers run in the periventricular area from the hypothalamic region caudalward to the brain stem, so that they are involved in lesions affecting the central gray. They terminate, among other regions, in the motor nucleus of VII, in the dorsal efferent nucleus of the vagus (not illustrated), in the nucleus ambiguus and in the medial reticular gray of the midbrain, pons and medulla. They end within the respiratory center (medial reticular gray) of the medulla and it is altogether probable that they reach the pneumotaxic center in the pons which stimulates the respiratory fields, increasing the respiratory rhythm.

In the respiratory center of the medulla they come into relationship with the descending pathways[1] of the ventrolateral reticulospinal tracts which distribute to the neurons supplying the diaphragm and the other muscles concerned with inspiration. Ordinarily the expiratory field serves to inhibit the inspiratory field periodically. However, in hysterical coughing or in excitement, expirations may be forced; then those connections of the expiratory center through the ventrolateral reticulospinal tract with the motor neurons supplying the muscles of expiration (abdominal muscles and internal intercostals) and the fascicles passing to the nucleus ambiguus (through which are innervated the abductors, the posterior cricothyroid muscles of the larynx) are important in expiration. Stim-

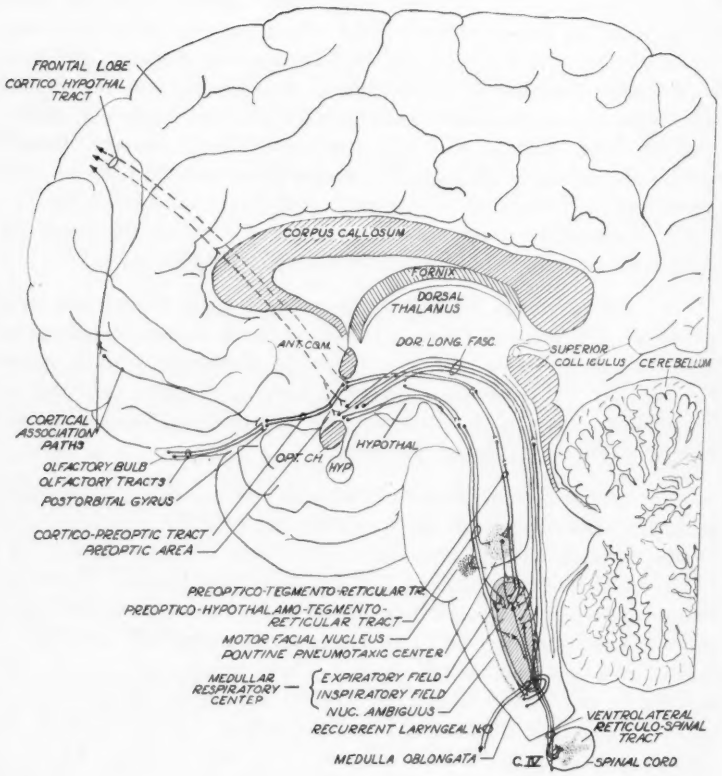

Fig. 1.

ulation of (1) the preoptico-hypothalamic transition area, or (2) the periventricular region of the midbrain through which the dorsal longitudinal fasciculus runs, produces an increase in respiratory rate and amplitude.

B. Other descending paths from the hypothalamus are concerned in increasing the respiratory rate. From the ventral part of the preoptic area and the anterior and dorsal regions of the hypothalamus, preoptico-hypothalamo-tegmental fascicles (preoptico-hypothalamo-tegmento-reticular tract) pass through the lateral hypothalamic area into the tegmentum of the midbrain near the region of emergence of the oculomotor roots. With or without a synapse in this region the system proceeds caudalward (probably as a chain

of neurons) to end in the appropriate motor nuclei (Fig. 1) and the pontine pneumotaxic center where, with and without relay, discharge is made to the medullar respiratory center. Here, the expiratory field rythmically but more rapidly inhibits the inspiratory field. Discharge to the motor centers of the cord makes possible increased respiration. This is part of the temperature regulating path.[7]

2. *The Neuron Path from the Cortex to the Centers in the Medulla and the Cord Inhibitory over Respiration.* (1) Corticopreoptic and corticohypothalamic[5] fibers extend to periventricular and posterior hypothalamic areas from which areas a pathway descends through the midbrain tegmentum with partial synapse in course to the pontine pneumotaxic center and to the medullar expiratory field. Impulses passing over this chain are inhibitory to these pontine and medullar centers, since stimulation in cortex[4] or midbrain (our own material) results in an arrest or, with lessened stimulus, a diminished amplitude or a slowing of respiration or both.

Meningitis.—Experimentally we are able to trace well-established neuron paths from the cerebral cortex to the centers in the medulla and cord which influence the rate of respiration. In the early stages of meningitis, and particularly frontal lobe abscess, we frequently observe slow, sighing respirations. The rate may be decreased to eight or ten a minute. This has been attributed to an irritation of the vagus nerves which inhibit respiration, and the pathological change has been assumed to be a purulent exudate on the brain stem in the region where the vagus nerves make their exit from the medulla. It has been stated also that increased intracranial pressure was occasionally the cause of irritation and that this likewise was exerted in and about the brain stem.

Autopsy findings in a number of my cases of meningitis, particularly those arising from nasal accessory sinus disease, were not consistent with this theory. It was frequently observed that respirations were slowed appreciably before there was evidence of increased intracranial pressure. Moreover, postmortem studies often revealed the presence of a thick exudate on the inferior surface of the cerebral hemispheres with little or no pathological change about the medulla and pons. This was invariably a disillusionment to me, when, during the course of the disease, we had ascribed the respiratory changes (first slow due to vagi stimulation and then rapid due to paralysis of the nerves) to inflammatory changes in the posterior cranial fossa.

Our recent studies on the brain of the monkey bring a fresh point of view and a new approach to the cause of respiratory changes in meningitis secondary to nasal accessory sinus disease. A more rational explanation for an early decrease in respiratory rate can be found in an irritation of the postorbital gyrus. This structure is superficially placed upon the inferior surface of the cerebral hemispheres and is accessible to an invasion of the meninges and brain from ethmoidal and sphenoidal sinus infections. It is undoubtedly the seat of a round cell infiltration in meningitis in this region and, when irritated, causes a definite slowing of the respiratory rate. By the same token, stimulation of the preoptico-hypothalamic transition area, a region slightly caudalward to the postorbital gyrus, produces an increase in respiratory rate and amplitude, a change usually observed in the terminal stages of meningitis or brain abscess. This structure is also superficially placed and accessible to an inflammation of the meninges and brain which has become extensive and deep seated. The implications therefore are quite obvious. These are the regions capable of governing the rate of respiration and because of their vulnerability to intracranial infections from the nasal accessory sinuses are in all probability responsible for the respiratory phenomena observed in meningitis and brain abscess arising from these sources. This explanation is more conformable to rationalistic principles than that which attributes the changes in the rate of respiration to a direct attack upon the vagus nerves at their emergence from the brain stem.

HICCOUGH

An example of a respiratory variation which may be due to peripheral or central lesions is the well-known hiccough. The hiccough is an inspiratory reflex of clonic type induced by sudden contraction of the diaphragm with the glottis partly or wholly closed. The sensory side of the arc in hiccoughs may be provided by the sensory fibers of the phrenic, the afferent fibers of the vagus or by other sensory neurons. In the diagram (Fig. 2) the sensory fibers of the vagus and the phrenic are shown. The sensory fibers of the vagus terminate in the nuclear gray of the fasciculus solitarius. From this nuclear gray intercalated neurons project the impulses on the inspiratory field of the respiratory center. From this field, neurons send their neuraxes to the cervical levels of the cord (C 3 to C 6) where they terminate around the motor neurons which provide the efferent fibers of the phrenic nerve. The descending neuraxes from the respiratory field to the spinal cord constitute the ventrolateral reticulospinal tract. The sensory fibers which come in

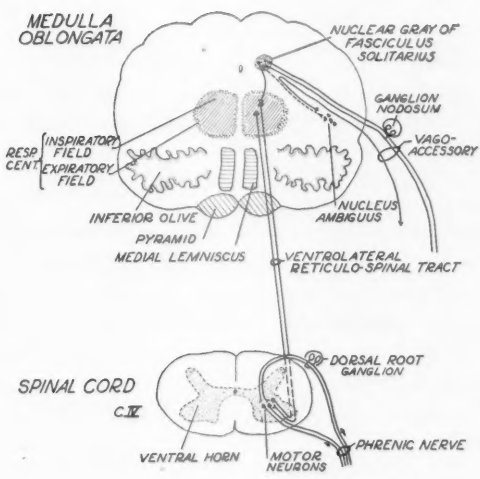

Fig. 2.

over the phrenic nerve send neuraxes into the ventral horn region to terminate directly around the neurons giving rise to the phrenic nerve.

This reflex may persist for only a short time (as in the intoxicated person) and have no clinical significance. It may be set up by peripheral stimulation of the sensory side of the arc, as in aortic aneurism or pleurisy of certain types, and may be sufficiently severe and long standing to warrant freezing, crushing, or section of the phrenic nerve. It may appear in tabes dorsalis where the dorsal root ganglia are irritated at cervical levels. In anterior poliomyelitis it may be a transitory symptom due to irritation of the ventral horn neurons of the cord at the level of the fourth cervical segment. This phase is usually followed rather promptly by a degeneration of the neurons supplying phrenic fibers to the diaphragm with a resultant paralysis of the muscle fibers on the side of involvement. It is to be noted that occasionally unilateral paralysis of the diaphragm causes difficulty in breathing (sometimes with recurrent attacks of cyanosis). Persistent hiccoughs are present in some cases of lethargic encephalitis, indeed are said at times to be the only symptom of this condition, and are thought to be the result of a myoclonus of the diaphragmatic muscles. They are observed occasionally in an epidemic form (epidemic singultus) in which the pathological changes

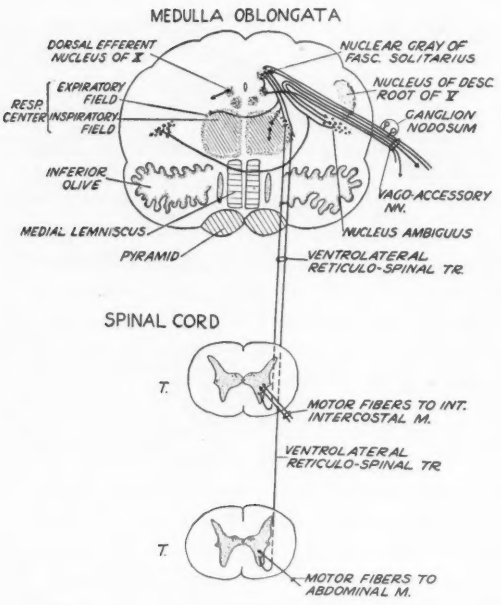

Fig. 3.

do not appear to be known. They may appear also in certain psychogenic states and a recognition of this fact is exceedingly important. There are definite central pathways for the transmission of emotional impulses and I shall deal more specifically with them in considering the mechanism of coughing. The point I wish to emphasize is that underlying so simple a modification of respiration as hiccoughs there may be a wide range of pathologic conditions.

COUGHING

Coughing provides another illustration of a behavior pattern which may be due to peripheral irritation of some part of the respiratory mechanism or to involvement of the central nervous system. Coughing is an expiratory reflex frequently associated with difficulty in breathing. The reflex pattern is illustrated in Fig. 3. The sensory fibers of the vagus nerve may be stimulated by irritating particles in the larynx. Such sensory fibers have their cells of origin in the nodose ganglion and their neuraxes terminate in the nuclear

gray of the fasciculus solitarius. From the nuclear gray of the fasciculus solitarius fibers pass to the expiratory field of the respiratory center. From this expiratory field the impulses pass over the ventrolateral reticulospinal tract to the motor neurons of the spinal cord which supply the muscles of expiration. From the expiratory field neurons also pass to the inspiratory field to inhibit inspiration. From the nuclear gray of the fasciculus solitarius crossed and uncrossed fibers pass to the nucleus ambiguus of the same and the opposite side. This nucleus provides fibers of the bulbar accessory nerve. Through this nerve abduction occurs in the larynx and the way is open for expiration. If the particle should have been drawn beyond the larynx into the trachea or bronchi, contraction of the smooth muscles of the bronchi may aid in expelling it. This smooth muscle is innervated by a two-neuron chain with the preganglionic neuron in the dorsal efferent nucleus of the vagus. They synapse with post-ganglionic neurons at the hilus of the lungs which terminate in the circular muscle of the bronchi.

On the efferent side of the arc coughing may result from pressure on the recurrent laryngeal nerve, as in aortic aneurism. It may appear from irritation of sensory ganglion cells, as in tabes dorsalis, or from irritating lesions in the nose and throat, or in children, in the mouth as an accompaniment of dentition. Involvement of efferent fibers of the superior laryngeal nerve may likewise produce coughing or interference with breathing.

Pertinent to this subject and worthy of perhaps more sympathetic understanding than has been accorded it is that type of reflex coughing which must be attributed to etiological factors of psychogenic origin. It should be recognized that there is a definite central mechanism (Fig. 1) for transmission of emotional impulses. We have all had the experience many times of consulting with the mother who tells us that her child has a chronic cough. He coughs day and night. He may sleep for a time when first put to bed but awakens after midnight much to the distress of the parents and coughs more or less for the remainder of the early morning hours. In the vast majority of these cases we look in vain for an organic etiological factor responsible for this symptom. There are no abnormalities in the larynx, the pharynx presents no pathological changes, the cough cannot be attributed to anything within the nose and radiographs of the chest are entirely negative.

I should like to emphasize that this symptom is frequently on an emotional basis. In fact, I am convinced that it is more often than not an expression of some strong feeling or impulse of a psycho-

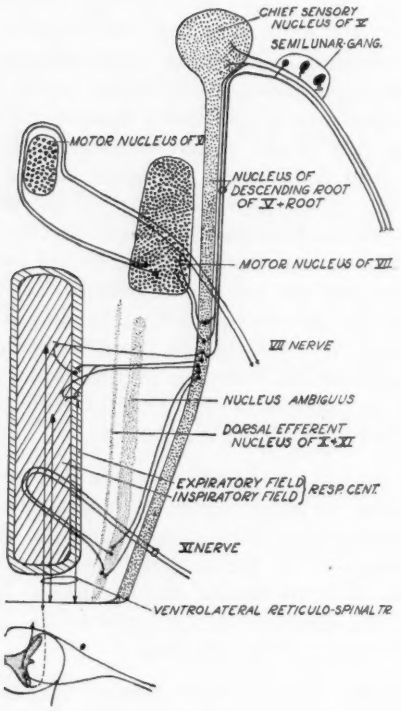

Fig. 4.

genic nature. Working in close cooperation with the psychiatrist in the care of a number of these patients, a specific pattern of parent-child relationship has been frequently revealed. There is an extreme dependence upon the mother. A feeling of frustration develops because the child is receiving too little parental love and attention. Rigid discipline, repression and the enforcement of vigorous rules of conduct rob the child of his security and leave him stranded in an environment which he considers devoid of parental love and affection. Coughing, therefore, often fulfills the child's purpose, namely, to bring one of the parents, usually the mother, to dispel his fears and give him solace and comfort. However, the cough is seldom voluntary. It is often produced while the child is sleeping or at least in a state of semiconsciousness. Some asthmatic patients

are the victims of a similar emotional conflict. It is another form of respiratory variation which is occasionally initiated and maintained by influences which are largely psychogenic in character.

Whether we realize it or not we are all influenced by factors extrinsic to ourselves. These influences of an emotional nature may act upon a mechanism of respiration which exists as an anatomical entity in the cerebral cortex. The pathways for the transmission of emotional impulses can be clearly defined and are capable of giving rise to a great variety of respiratory changes, among which we may find the cough of the insecure child or the emotional release of the asthmatic.

SNEEZING

Sneezing is a modified respiratory reflex in which a long inspiration is followed by a short sharp expiration. The reflex pattern is indicated in Fig. 4. Irritating substances may set up nervous impulses in the peripheral fibers of the Vth nerve. These nerve fibers have their cells of origin in the semilunar ganglion and the neuraxes terminate in the chief sensory nucleus of V for tactile, especially discriminating tactile, and in the nucleus of the descending root of V for pain, temperature and general tactile. From this nucleus connections are made with the motor nucleus of the VIIth nerve, with the respiratory fields and with the nucleus ambiguus. The connections with the motor nucleus of VII from which motor fibers pass to the muscles of the nose complete the reflex arc, the sensory side of which is provided by the root fibers of V and the motor fibers by the root of VII. If any part of this reflex arc is destroyed (as for example, the peripheral fibers of the trigeminal nerve) the reflex arc will not function and the nasal openings will not widen with the passage of air into the nostrils.

The nucleus of the descending root of V is connected with the inspiratory portion of the respiratory center. From this inspiratory field connections are made through the ventrolateral reticulospinal tract to the motor centers of the cord which initiate inspiration. Thus the long inspiration is produced which precedes sneezing. The muscles of expiration are stretched as the muscles of inspiration contract. From these stretched muscles sensory impulses are relayed through a series of neuron arcs to the expiratory field of the medulla. The connections from the expiratory to the inspiratory field serve as a pathway for inhibition of the inspiration. Where the expiration is violent, as in sneezing, connections are made from the expiratory field to the motor centers of the cord innervating muscles of expiration through the ventrolateral reticulospinal tract.

A phenomenon in this connection which has excited my interest and curiosity for a number of years is that observed in the individual who has experienced a successful submucous resection of the septum for the relief of nasal obstruction but continues to complain bitterly of an inability to breathe through the nose. The nasal cavities are wide open. There is a large volume of air traversing the nasal chambers with each respiration but the patient vigorously defends his contention that he still cannot breathe adequately.

We might conclude (and I suspect properly in some cases) that this is a hysterical manifestation. Yet I doubt seriously that this explanation will suffice to explain the factors in the majority of patients which give rise to this complaint. There is perhaps a more rational reason found in an evaluation of organic factors which have altered the respiratory reflex arc described for the act of sneezing. For example, it has been demonstrated frequently in our laboratory of physiology that one may regulate the rate of respirations within certain limits by stimulation of the nasal mucous membrane. The respiratory rate may be altered within a range of from 14 to 24 respirations per minute in a tracheotomized dog by blowing air from a bellows through the nasal cavities. The experiment clearly indicates that one may bring an extraneous influence into play which governs the rate of respiration to a limited extent.

Scientists familiar with this field will tell us therefore that something has altered the reflex respiratory arc in those patients with wide open noses who complain of impaired breathing after a submucous resection of the septum—that we have injured or in some manner altered the sensory side of the arc provided by the fibers of the trigeminal nerve. Although the nerve supply to the nasal mucosa is wide spread and a considerable part of it outside the field of the submucous operation, there may occur, nevertheless, sufficient damage to the sensory distribution to alter the pathways which furnish the impulses of respiration. A considerable number of my patients during the past 30 years have complained of a loss of the sense of smell after a submucous resection of the septum. While this has usually been temporary, there have been instances when the sense of smell remained permanently impaired. In several cases it has seemed plausible to explain this disturbance as injury of the middle turbinate or the mucous membrane of the septum adjacent to this structure; there have been patients in whom adhesions between the middle turbinate and the septum were obviously the offending factor but in the vast majority of my patients the mucous membrane of the nose appeared normal and entirely de-

void of any pathological change which would imply damage to the end organs or fibers of the olfactory nerves.

If it is possible to impair the sense of smell without subsequent evidence of any gross pathological changes in the mucous membrane of the nose, is it not therefore feasible to expect that the submucous resection of the septum might do similar damage to the end organs of the trigeminal distribution or some of its important fibers. The point to be emphasized is that, in some patients who complain of difficult breathing in the presence of wide open nasal cavities after the submucous resection of the septum, there is perhaps a definite organic basis for this phenomenon arising from a disturbance in the sensory innervation of the nasal mucous membrane. Perhaps, too, in some cases the olfactory sense plays a part because we know that it is directly connected with the central emotional system which exerts an important influence upon the respiratory centers in the brain stem. As further proof of this theory which ascribes this phenomenon to some change in the nasal respiratory reflex arc is the fact that these patients usually recover spontaneously when sufficient time has elapsed to permit regeneration of trigeminal fibers.

CONCLUSIONS

1. As the title indicates an effort has been made to give due consideration to the peripheral and central pathways over which the impulses of respiration are transmitted. Special emphasis is given the central tracts in describing certain common respiratory variations.

2. The absence of pathological changes about the brain stem in individuals who had died of meningitis gave rise to doubt that direct stimulation of the vagus nerves and later paralysis of them were responsible for the respective slowing and acceleration of respiration. The inquiry into a more rational explanation for these changes arose particularly from autopsy findings in meningitis secondary to nasal accessory sinus disease where the exudate was largely confined to the surfaces of the brain. Laboratory experimentation on monkeys demonstrates well-established areas in the cerebral cortex which when stimulated either slow or accelerate respiration. Autopsy material from patients who died of the type of meningitis above described demonstrated inflammatory involvement of these regions.

3. While the text probes only superficially into the emotional factors responsible for respiratory variations such as hiccough, sneezing, and coughing, it treats specifically of the central nervous apparatus along which emotional impulses are carried. A knowledge

of these pathways and a better understanding of parent-child relationships may widen our knowledge of the psychology of cough and asthma.

4. In discussing the reflex arc of sneezing, an effort is made to offer an explanation on an organic basis for the occasional complaint of nasal obstruction after a submucous resection of the septum in a patient who has wide open nasal cavities. Although the picture is complicated, a disturbance of the pathways of the reflex arc is described which may properly explain this phenomenon.

5. Finally this initial inquiry into the nervous mechanism of respiration and its common variations leads one to the conviction that much more work needs to be done if we are to achieve a thorough survey of this subject. The one generalization that may be derived from this discussion is that the whole mechanism of respiration is a complicated one governed by a reflex arc and an equally important central nervous system component susceptible to emotional stimulation. In looking for the cause of respiratory changes we must remember not to confine our efforts to an investigation of organs supplied by the peripheral nerves. A knowledge of the central nervous system mechanism and its connections with the peripheral nerve distributions may perhaps help us as laryngologists to think rather than feel our way toward a better understanding of respiratory abnormalities.

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XXVIII

DISCUSSION OF PRESENT METHODS FOR TESTING AUDITORY FUNCTION

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My subject indicates that this presentation will offer a critical discussion of the methods used to make diagnoses and to measure the hearing of acoustically handicapped patients.

There are sufficient errors in our present diagnostic methods and tests used in the measurement of hearing to warrant accurate reevaluation. A wide discrepancy in the reported results of the fenestration operation stimulated my interest in this subject. Since the surgeons who have reported their results following the fenestration operation enjoy a reputation in our specialty which insures veracity and since the surgical ability and technique of those surgeons is of insufficient variance to account for the great discrepancy in the percentages they have reported, I believed only one place remained which might offer logical causes for such a wide variation in reports. As a group, this source includes:

1. Our deductions from traditional but unproved experiments on which our methods of diagnosis have been founded.

2. The variability of the human element on the part of our patients and ourselves as examiners.

3. Mechanical errors in instruments which we use to aid us in our diagnosis and measurement.

Diagnosis on the Authority of Unproved Theories.—First, let us consider the integrity of our commonly accepted diagnostic methods plus the authority for our arbitrary opinions. I have used Kerrison's[1] book on otology as a text for teaching over a period of many years. His book still stands as one of the most substantial, truthful and reliable books of our time. He starts his chapter on "Functional Examination of the Cochlear Apparatus" by stating, "To the aver-

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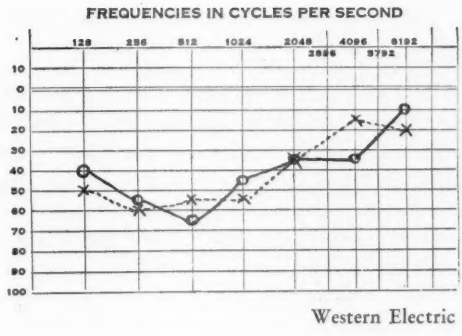
age practitioner of medicine, the functional examination of the ears is an unknown act." He was charitable in using the word "practitioner" and not "otologist." It may be the lack of knowledge and, perhaps, interest on the part of some of us which leads us to accept, without question, impressive theories and awe-inspiring complicated instruments as being inviolate.

Again quoting from Kerrison,[1] "The explanation of the increase in hearing by bone conduction in tympanic diseases is not altogether clear." He quotes Bezold's hypothesis and comments that while not wholly convincing, it is as satisfactory as any advanced. In fact, nothing has been proposed since Bezold's hypothesis to change Kerrison's frank phrase and it remains today not "altogether clear." Yet, we make positive diagnoses and, on the questionable authority of these diagnoses, we advise the most difficult of all operations. We treat Bezold's theory as if it were a fact.

Once more, I will quote from Kerrison.[1] Under "Physiology of Sound", he states, "It may be well to acknowledge that the modern conception of the physiology of sound perception is based largely upon hypothesis and only to a limited extent upon demonstrated facts." In discussing Helmholtz's theory of tone analysis he again states, "At best, it is a hypothesis."

As an example of a commonly accepted fact born of an erroneous interpretation, I will submit Ewald's experiment in which he tried to demonstrate the stimulation of the cristae by fluid movement. You may have the same feeling of aversion that I experienced when questioning the deductions of Ewald. These early experiments have unfortunately acquired a sacred air because of their unquestioned acceptance over a long period.

The references to Ewald's work are all so similar that the selection of any one of them will serve my purpose. The textbook, *Physiology of the Vestibular Apparatus*, by Camis and Creed[2] states, "The procedure adopted by Ewald after some time consisted in fixing a metal cylinder 1.4 mm. in diameter and 9 mm. long, over a small hole previously made in the bony wall of a canal by cementing it to the bone. In the cylinder would run a piston of about 1 mm. in diameter, one end of which was tapered to 0.35 mm. and served to compress the membranous canal. The piston's movements were controlled with the aid of a rubber bulb connected to the hollow cylinder through a rubber tube. By means of this pneumatic hammer, as it was called by the inventor, it is possible to compress and decompress a point on a membranous canal, so causing a current of endolymph that at first flows away from the point compressed



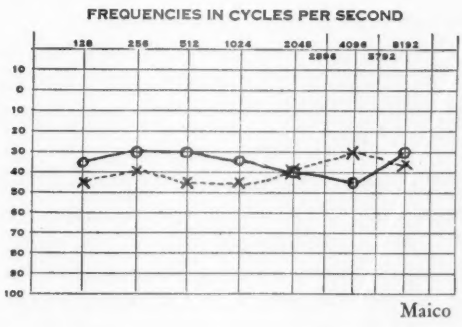


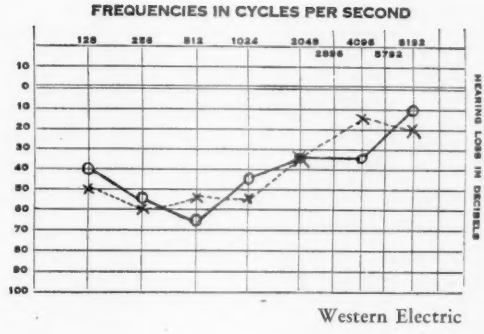
Fig. 1.—Audiograms made on the same patient by the same operator with two different audiometers both on the morning of November 5, 1947.

and then flows back to it." A footnote here reads, "The movement of endolymph would inevitably occur in two opposite directions, viz., towards the ampullary end and towards the simple end of the canal whenever a point near the middle of the arc is compressed." But in order to obtain movement solely towards the ampulla, Ewald plugged the other arm of the canal. Movement towards the plugged end being thus excluded, it was quite certain that the only effect of the compression was excitation of the ampulla.

Several features of this experiment may be questioned. The endolymphatic system is a closed system. There is no inlet or outlet except the theoretical stria vascularis of the scala media as a place where the endolymph fluid is formed and the endolymphatic sac which acts as a filter for the escape of endolymphatic fluid. The only opening to the perilymphatic space is the aquaeductus cochleae. The ductus endolymphaticus closes another bony canal housing that structure. The nerve end organs or cristae are entirely enclosed in the lumen of the ampullae which are the dilated ends of the semicircular canal. They are inside this closed endolymphatic system. Any outside force exerted on the perilymph could not cause a motion of fluid which would stimulate the cristae in the closed endolymphatic system.

Ewald's observations on the effect of labyrinth stimulation were probably correct, but his conclusions regarding the cause were undoubtedly wrong. According to the physical law regarding pressure on fluid in a closed space, his deductions were also wrong. The pressure is exerted equally in all directions; therefore, there would be no fluid movement. Other experiments need careful study to determine their soundness.

Human Discrepancies (Patient and Doctor).—We have placed the human element high on the list of potential errors. Some of our supposed "cures" have been the result of a psychological gain in the patient's ability to hear following "treatment with emphasis." I say psychological gain because no actual improvement in hearing can be demonstrated by any of our testing methods. The person with impaired hearing may be despondent during a test if this test follows numerous unsuccessful visits to otologists. Just another test may elicit a lethargic response on the part of the discouraged patient. When this same patient hears of a new miracle worker and then is subjected to an impressive emotional experience during the treatment, this patient may be alert and subconsciously concentrate during the next test. The result may be a ten or twenty decibel increase in hearing. This temporary increase will fade as



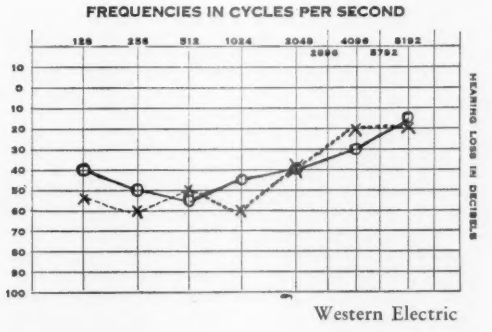


Fig. 2.—Two audiograms made at separate times on November 5, 1947, on the same patient, by two different operators, using the same audiometer. First test by R.B.; second by M.N.

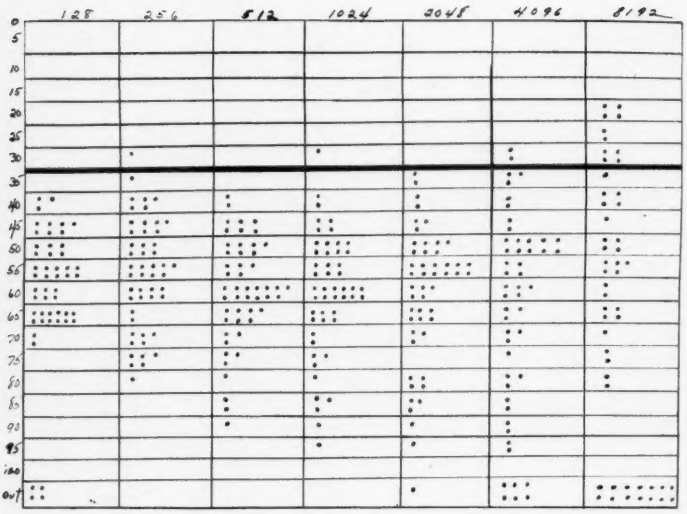


Fig. 3.—Scattergram of hearing of 50 patients in Group I before fenestration operation.

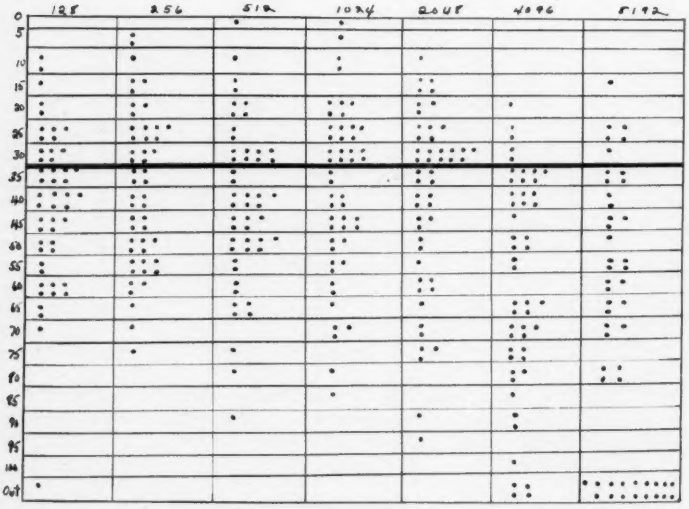


Fig. 4.—Scattergram of hearing of 50 patients in Group I one month postoperatively.

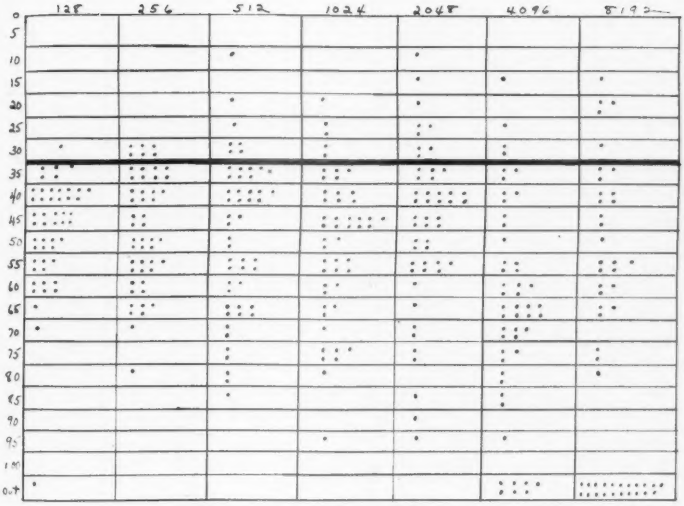


Fig. 5.—Scattergram of hearing of 50 patients in Group I one year postoperatively.

the patient's hopes again fall. We have all seen this happen to patients following various electrical treatments or surgery.

Patient fatigue is an important factor in the accuracy of testing. One patient in this series who was of normal emotional status was unable to continue the repeated tests because of fatigue. If a patient is tested while fatigued in the late afternoon and a second test is done the following morning after he has rested, there may be a substantial increase in his hearing. The examiner's enthusiasm to help the patient may lead him to unconsciously vary his tests with his mood.

Mechanical Errors in Instruments.—Instruments used as aids to diagnosis and used in the estimation of hearing ability can be divided in two eras, the audiometer and the pre-audiometer. In some offices the pure tone audiometer serves as the sole arbiter of diagnosis and degree of gain or loss in hearing. It is viewed with reverence and its results are accepted without question. The contrast between the high precision of the instrument and the gross errors inherent in its use would be the subject of ridicule, were not

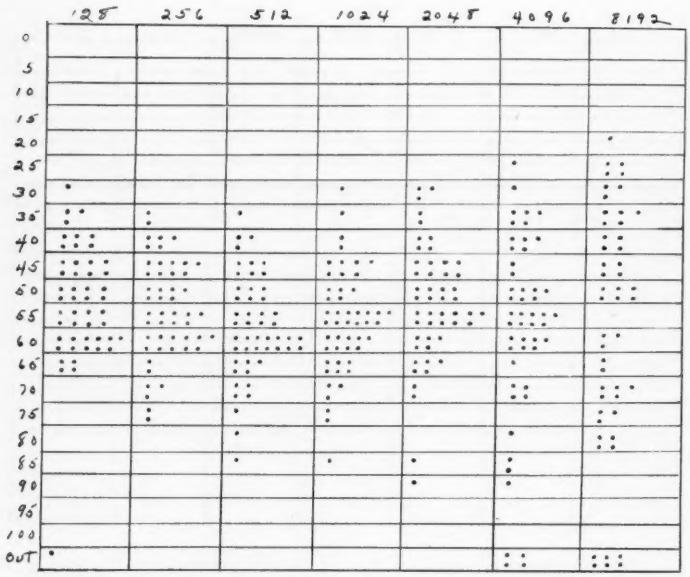


Fig. 6.—Scattergram of hearing of 50 patients in Group II before fenestration operation.

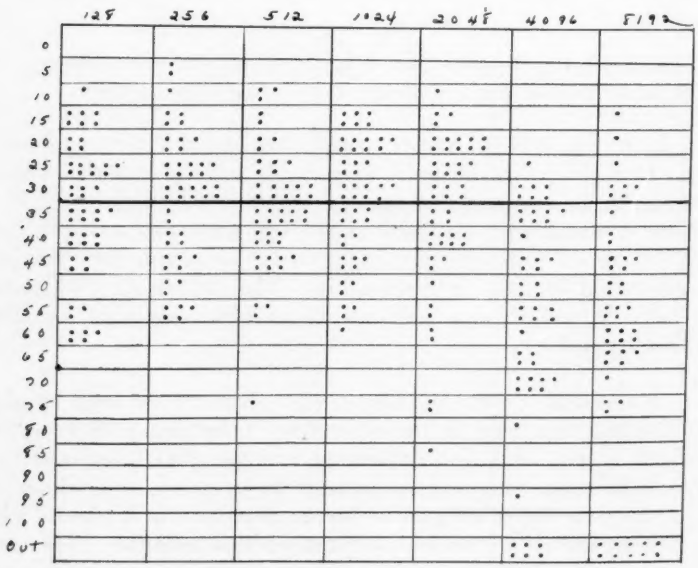


Fig. 7.—Scattergram of hearing of 50 patients in Group II one month postoperativeliy.

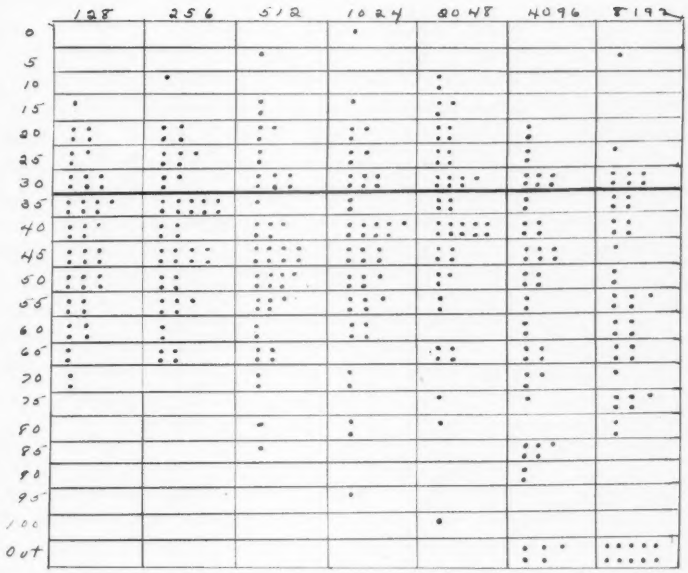


Fig. 8.—Scattergram of hearing of 50 patients in Group II one year postoperatively.

its position in the world of otology so sacrosanct. It resembles the human in that it has its own concealed faults.

Figure 1 shows two audiograms taken on the same patient by the same technician at the same time under the same conditions. Both audiometers had been returned to us by their manufacturers as being in "perfect" condition. The waiting time for the return of these repaired audiometers was never less than one month. There is no way in which the average otologist can tell whether or not his audiometer is functioning as it should. Audiometers do vary widely in their accuracy.

The next experiment shows two audiograms taken under similar conditions but with the same audiometer and different operators (Fig. 2). One was an audiologist and the other, an otologist. We have repeated this test often enough to prove to ourselves that honest people do not vary to any important extent in their testing.

The preceding tests were done to obtain estimates of the patient's ability to hear pure tones by air conduction.

FORK TESTS

	B.C.		A.C.	
128				R
				L
256				R
				L
512				R
				L
1024	25"	15" >	10"	R
	10"	< 20"	30"	L
2048				R
				L
WEBER TEST	R		L	

Fig. 9.—Record of relation of air to bone conduction and comparison of the two ears.

In testing bone conduction, I have found the audiometer to be too unreliable for me to risk important decisions on the results. Exceptions are granted in the case of a few large institutions where there is a constantly available, trained physicist. I always record audiometric bone conduction, but I place no confidence in that record.

Further, I make no important decisions that depend on a single audiometric record. If surgery is to be considered, I make no decisions on less than three audiometric recordings *plus* tuning fork examination. This statement may sound dogmatic, but I am sure time will confirm its wisdom.

It seems a proper place to suggest methods which may minimize errors and render reporting more reasonable. Averages taken of many tests under as near standard conditions as possible tend to neutralize the unusual recording and make it more noticeable by its variance.

I have accepted the 30-decibel level as the level of useful hearing for the three pure tones in the conversational range, 512, 1024, and 2048. At the same time I recognize the difficulties in making comprehensive reports based on the average of these three tones at this level. I suggest we report actual gains or losses in decibels for

each pure tone in the conversational range. Determine average results from monthly audiograms taken on days which shall be designated thirty days in advance for a period of one year. The year of recording for patients who have had fenestration operations should start on the anniversary of the day on which the operation was done. The object of this method is to simplify our records for comparison and to neutralize some of the causes for error which I have already mentioned. The scattergram method of doing these recordings is illuminating. The first scattergram shown is taken on a series of patients (the first 50 done by more than one surgeon) preoperatively (Fig. 3). The results from the same series one month postoperatively are shown in Fig. 4 and one year postoperatively in Fig. 5. The second series of 50 patients (operated on by the same group with an additional surgeon) is recorded in the same manner (Figs. 6-8).

It is obvious that the greatest improvement in hearing is during the first month. The first fifty operations were done by surgeons who had all performed less than 50 operations, but surgeons were also included in the second group who had performed less than 50. The second series is better than the first. I believe the accomplishments of the average good surgeon working under good average conditions should be reported. Experience seems to improve results.

The speech audiometer must be mentioned because of the recent advances made in this method of testing. My experience has been that the speech audiometer closely parallels the results obtained on the pure tone audiometer with the exception of a few cases in which they differ so widely that I believe there is a significance in the variation. Certainly the speech audiometer is not generally available today.

In discussing the elimination of errors I am somewhat reactionary. I still depend upon tuning fork tests as the final court of appeal when the most reliable information is desired, nor will I recommend any surgical procedure without the information I can get from their use. The most useful tuning forks are the 512, 1024, and 2048 double vibrations a second.

In using the tuning forks to determine the relation of air to bone conduction and the comparison of the two ears, I have used a method which gives me reliable information without masking. For practical purposes, this method gives useful information but may fail when the patient has one normal hearing ear and one profoundly deaf ear.

The 512, 1024, and 2048 d.v. tuning forks are used in this test. A record is made of the total time (with interrupted testing

periods) the three forks are heard by air conduction and of the total time they are heard by bone conduction. I then take the lesser time to again test the differential (Fig. 9). For example, the 1024 fork is heard 25 seconds by bone and 10 seconds by air. The bone conduction time is 15 seconds longer than the air. The fork is then timed by air conduction and when the sound ceases, it is placed on the bone. The differential should again be 15 seconds. With this method the relative ability to hear is registered and the intensity with which the fork is originally activated is unimportant. Since the patient has ceased to hear the fork by air, no masking is necessary. The reverse is also true. The same procedure is done on the second ear. For example, the air conduction is 30 seconds and the bone conduction is 10 seconds. The differential is 20 seconds longer by air than bone. The Weber test should be referred to the second ear tested and in both instances the Rinne is negative. The phenomenon of bone conduction needs a more satisfactory explanation than any advanced thus far.

Tuning forks may be calibrated by sending them to the United States Bureau of Standards, and once they are calibrated, they continue as a permanently known factor. Uncalibrated forks vary considerably.

SUMMARY

Repeated tests by audiometer and tuning forks tend to reduce the discrepancies caused by errors.

Some of our errors in diagnosis are caused by our acceptance of time-honored, unproved theories as facts. A meticulous review of all experiments on which our present diagnostic methods are based should be a continuing effort.

More reliable methods of diagnosis are urgently needed.

The effort to produce high precision instruments for testing hearing has been largely wasted because of gross errors on the part of fundamental information, mechanical imperfections, human response, and technique of examination. I wish to stress once more that I am speaking particularly of the average doctor and institution, not of the highly developed acoustic centers with their excellent facilities and most capable staffs.

In this work I have been assisted by Dr. Richard J. Bellucci, Mrs. Mary Whitehurst, Consultant Audiologist, and Mrs. Marjorie Nerenberg, Audiologist, of the Manhattan Eye, Ear, and Throat Hospital.

121 EAST 60TH STREET.

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2. Camis and Creed: Physiology of the Vestibular Apparatus, Oxford University Press, 1930, pp. 71-72.

XXIX

THE VOLUME OF THE BRONCHIAL TREE AT VARIOUS LEVELS AND ITS POSSIBLE PHYSIOLOGIC SIGNIFICANCE

A. C. Hilding, M.D., and David Hilding

Duluth, Minn.

We consider the nose to be an efficient air cleanser, and so it is. It removes the dust and bacteria which are in suspension, and at the same time humidifies the air. Despite this, some particles do get through to the alveoli, as is indicated by the disease of silicosis and by the black spots one sees on the pleura of the lungs in any adult subject at necropsy. This brings up the question as to whether or not there is any other air cleanser lower in the tract. Proetz[1] states that there is, but does not enlarge upon the subject. A laryngectomized person gets along very well without a nose. Nature seems to have a safeguard, or substitute, against the loss of most other organs. What is the subglottic substitute for the nose?

One of us had occasion a few years ago to make some preliminary measurements on the bronchial tree of a dog. These incomplete measurements gave the impression that there might be a constriction of the total volume of the respiratory tract a short distance below the bifurcation. We looked up the volumetric figures of the human bronchial tree in the literature but were unable to find anything except on the tiny microscopic branches[2, 3] (Fig. 1). dePablo[4] has worked out the architecture of the bronchial tree very thoroughly but gives no comparative volumetric figures. A number of internists, and physiologists, and anatomists were asked if they knew what the measurements might be. None could give any specific information, but all were of the opinion that the volume of the tract increased progressively with the branching in much the same way as does the circulatory system.

Proetz, in his book on "Physiology of the Nose",[1] shows some illustrations from experiments in which dust-laden air was passed

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From the Department of Ophthalmology & Otolaryngology, University of Minnesota.

Read before the American Laryngological Association, Hot Springs, Va., April 14, 1948.

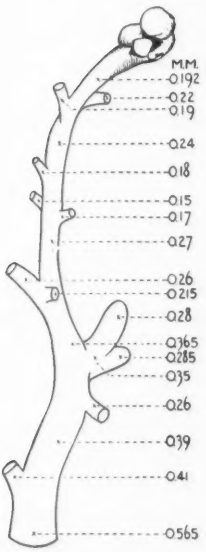


Fig. 1.—Diagram of a reconstruction of a bronchiolus and the structures between taken from W. S. Miller.[2] According to these measurements, there is a ninefold increase in the total volume of the air passages between the bronchiolus and the alveolar duct. Beyond the latter there is a still greater increase in volume.

through glass tubes of various forms. He found that if there was constriction in such a tube, the dust would be deposited just beyond the constriction. It also settled just beyond any bends or turns in the tube. The possibility occurred to us that if there were a constriction or isthmus in the bronchial tree, it might be purposeful. It might act to deposit such dust as the air entering the trachea might carry with it upon the lining mucous blanket, which is, of course, carried away by ciliary action. It was decided to make some measurements of the bronchial tree of man.

The material used consisted of a few specimens of lungs, or portions of lungs, removed at necropsy. One was the entire tract from a stillborn infant, and four were from adult males whose ages were respectively: 34, 44, 80, and 83. The bronchial trees of the left upper and left lower lobes of the newborn were measured; the left

lower lobe of the 34-year-old male, the right middle and right lower lobes of the 44-year-old male, and the left lower lobe of the 83-year-old male. The entire tract of the 80-year-old male was obtained but the data was discarded because it was found to be unsatisfactory. Fifty-seven bronchi were measured in the left lung of the newborn, and in the adults, from 96 to 276 in each lobe.

It was intended at first to make corrosion specimens by filling the respiratory tract with a light-weight resin and then dissolving away the tissue. Thus far, we have been unable to obtain the resin. An attempt was then made to fill the entire respiratory tract of the 80-year-old male with gelatin and section the gelatin at intervals of 1 cm. This method proved to be impractical and the specimen was discarded. In the remaining specimens the entire bronchial tree was laid open by slitting it posteriorly. It took some little practice to cut the lung tissue in such a way as to avoid cutting across the bronchi. When a lobe has been dissected out in this manner, the lung tissue is cut into thin slices which lie in anteroposterior planes. If any considerable portion of lung remains intact, then some branches have been missed. Each branch was followed as far as possible with probe and scissors. Despite great care some may well have been missed. Measurements were made at points just beyond each division. The bronchi were grouped according to the number of divisions through which they had passed.

The measurements were made across the slit bronchi as they lay flattened out in the form of bands or ribbons by means of a small millimeter rule. As the measurements were made, the figures were recorded in a diagram similar to that used for the generations in family trees. About 650 measurements were made. These measurements corresponded to the circumference of the bronchi during full distension. The cross-sectional areas were calculated from the circumferences.

This method is subject to a number of sources of error. The trachea and primary bronchi are not round and, using measurements as obtained, the figures for cross-sectional area would represent perfectly round tubes. Another source of error is the distensibility of the membranous bronchi. When they have been slit open, it is very easy to stretch the band of mucosa in such a way as to make the circumference seem larger than it truly is. Therefore, the measurements of the trachea and bronchi which have cartilaginous arches, or rings, in their walls yielded values which were greater than the actual cross-sectional area during life. On the other hand, the membranous tubes in life are not fully distended in ordinary respiration.[7] There-

fore, these values are also probably considerably exaggerated since in each case these measurements would be those of maximum distension. (The criticism of casts made from heavy Wood's metal is that the delicate membranous tubes become greatly distended.) During the respiratory cycle the membranous bronchi and bronchioles vary greatly in diameter, whereas the variations in the bronchi and trachea, which have rings, is comparatively slight during the respiratory cycle. At best the figures obtained are only approximations.

Another source of error was the irregularity of branching. In some regions it seemed dichotomous, but more often monopodous or a mixture of dichotomous and monopodous. Occasionally the branching was found to be trichotomous. This irregularity in branching made it difficult to classify the tubes into groups which would represent comparative progression along the tract. At first it was thought that all branches should be measured at regular intervals at a given number of centimeters below the bifurcation. This, however, did not prove to be feasible. Some would branch two or three times before others had branched at all. Some would go through three or four divisions, whereas others would go through eight or ten.

The figures of the cross-sectional areas of the various bronchi were added together in order to construct a single tube. In so doing it was necessary to use some of the cross-sectional areas at several different levels more than once. For instance, if one bronchus, coming off at a right angle high in a lobe, went through just two or three divisions to reach the minimum where measurements were possible and the other branches, going deeper into the lung, went through many divisions, it was necessary, in order to indicate the full volume of the tract, to include the measurements of the short branches together with the longer branches at each successive lower level.

Measurements were made after each branching or division, and each branch was followed out as far as it was possible to measure by this method. The measurements were all grouped according to the order of branching. For instance, all measurements just beyond the fifth division in all the different branches were grouped together and the cross-sectional area of each tube calculated. In order to give a composite picture of the volume of the entire tract, all of the cross-sectional areas of all of the tubes at each level were added together. From these values composite diameters were calculated and a single composite tube diagrammed. Also, all of the measure-

ments which were made were added together to give composite figures of the circumference.

RESULTS

It was found practically at the outset that there was no such constriction as the preliminary measurements on the dog seemed to indicate. There were indeed many minor irregularities, such as the formation of a sort of hood at the beginning of a bifurcation where the cross-sectional area was greater than either just above or below this point. There were also many bends and turns but, as far as could be determined, there was nothing that would indicate the use of eddies for the purpose of cleansing the air.

It also became apparent that there was no marked progressive uniform increase in the total volume between the trachea and the small bronchi as seems to be the general impression. In most instances there was a small increase, about 50 per cent to 100 per cent, and there was a corresponding small increase in the calculated composite diameter, up to 40 per cent. There were some small irregularities, probably due to error in the method of measurement, but generally the composite diameters were surprisingly consistent and uniform.

When a single tube is constructed from these calculated diameters, representing the total cross-sectional area of the bronchial tree at different levels from the trachea to the small bronchi, it is found to have a surprisingly uniform caliber (Figs. 2-6). (The length of the diagrammatic tube has no significance.) A tube constructed from the composite circumferences, on the other hand, opens out like a funnel and would represent the area of ciliated mucosa with which the air must come in contact. This great widening is due, of course, to the fact that the total circumference of many small tubes is much greater than that of a single tube of equal volume. The number of tubes in the baby increased from one (the trachea) to 35 small measurable tubes in the periphery of the left lung. Single lobes were measured in the adult specimens. The increase in these varied from one to 35 to one to 96.

DISCUSSION

The principle of air eddies probably is used in the upper part of the nose to bring air into the olfactory fissure, but it is not important in the cleansing of air. On the other hand, more direct impingement is probably much more effective, and it occurs more

or less all through the tract at the irregularities, turns, and branchings. The conception that the total composite volume of the respiratory tract increases uniformly and progressively in volume, as it does in the blood vessels, seems to be inaccurate. The figures for the blood vessels given by Glasser[5] indicates that the volume of the arterial tree increases fourfold in passing from the aorta to the primary branches, which are about 40 in number. The volume of the arterioles is about 150 times, and the volume of the capillaries 700 to 800 times, that of the aorta. This is more than the measurements found in the bronchial tree where the increase from the larynx to the smallest tubes which we measured is only from 50 to 100 per cent.

The conditions in the two systems are, however, very different. In the blood vessels a rather heavy fluid is passing under pressure at a fairly high velocity. Friction increases very rapidly as the diameter of the vessels is reduced and is very great indeed in the microscopic vessels. If there were no increase in total volume, the work of the heart would be increased tremendously. Moreover, the flow in the vascular system is all in one direction. There are no stagnant pools of blood in the arterial tree. The material passing in the bronchial tree is a gas and the friction factor is very much less. This is not only because of the difference in nature of the two materials (blood and air) but also because the velocity of the latter is comparatively low.* In ordinary quiet respiration the flow down the trachea is calculated to be about one meter per second, and perhaps half that rate in the smaller tubes. There is another factor which is entirely different from the condition found in the circulatory system; namely, the air does not pass continuously through the tubes, but flows first in one direction and then comes to a stop, and then flows back again. This means that at the end of inspiration the bronchial tree is left full of fresh air, and at the end of expiration is left full of alveolar air. In other words, the volume of the tracheobronchial tree which is about 150 cc. is dead space. We take in about 500 cc. of air with each breath in quiet respiration but the first 150 cc. which passes into the alveoli is not fresh air. It is the last 150 cc. to have been expelled at the last previous expiration. Therefore, we get 350 cc. of fresh air with each inspiration. If the volume of the respiratory tract were increased tenfold (a modest increase compared to the circulatory system), then the dead space would be 1500 cc. and in order to get 350 cc. of fresh air we would have to inhale 1850 cc. with each breath. This would seem

*The velocity of the blood in the aorta has never been measured accurately. It may be that the velocity is not as great as is generally supposed.

to be both uneconomical and unnecessary. Such an increase in volume would require a larger chest cavity.

On the other hand, somewhere between the smallest tubes which we have measured and the alveoli there is a very large increase in volume. Miller[6] has shown this in his book in an illustration of a reconstruction of the fine air passages between the bronchiolus and the alveolus. There are other data[3] indicating that there is a substantial increase in total volume in these small air tubes of less than 0.5 mm. in diameter. This would mean that there is a very large number of these small tubes which together would form an effective filter. One trachea among these specimens was very carefully measured, and the cross-sectional areas are found to be about 340 square mm. If an area of 340 sq. mm. is divided into 1 mm. tubes, 430 tubes will be required. If the tubes are 0.5 mm. in diameter, it will take 1730 of them to make up a cross-sectional area of 340 sq. mm. If the tubes are 0.35 mm. in diameter, which is about the size of the terminal bronchioles, it will take 3700 to make up a total cross-sectional area of 340 sq. mm.—this without allowing for any increase in any total volume at all below the trachea. We have found that the volume does about double. In addition, according to Miller's measurements, there is a volumetric increase of five to tenfold in the passages less than 0.5 mm. in diameter before the alveolar ducts are reached. The greatest increase in volume of the whole tract apparently takes place between the alveolar ducts and the alveoli. The ciliated mucous membrane extends down through the whole tree as far as the respiratory bronchioles. Here, where the alveoli begin to appear, the cilia gradually disappear. The terminal bronchioles, however, are well ciliated. One illustration in Miller's book pictures a bronchiole 0.35 mm. in diameter, and the cilia are very well formed. Considering bronchioles 0.35 mm. in diameter and making calculations from the data which we have, there would be at least 15,000 to 70,000 of them, and possibly many more. These would constitute a very efficient filter since each of the bronchioles is lined with a blanket of mucus. Assuming that there are 25,000 such tubes, their total cross-sectional area would be 2,250 sq. mm., and their composite circumference about 26 meters. That would mean that the 500 cc. of tidal air, which we pass back and forth in quiet respiration, would be divided up into 25,000 small jets and would be distributed over a mucous blanket 26 meters in width. The greatest possible distance between a dust particle and the mucous blanket would be 1/6 mm. The rate of flow would be not over 15 cm. per second. Such an arrangement would furnish a very effective filter and a last chance depository for

dust and suspended matter before the cilia cease. When the respiratory bronchioles are reached, the epithelium changes to a nonciliated form.

For this air filter made up of bronchioles, may we suggest the name of "bronchiolar filter"?

The protection of the delicate alveolar lining from damage by inspired air is impressive—first, the efficient cleansing, warming, and humidifying action of the nose; then further cleansing and warming of the air as it flows down the bronchial tree; final cleansing in the bronchiolar filter where it passes over several meters of ciliated mucosa. There is further protection in the progressive reduction in velocity of flow in the small passages from one meter in the trachea to probably one or two mm. per second as it enters the alveoli. In addition to these measures, 350 cc. of fresh air in quiet breathing is preceded by 150 cc. of air which has already been in the alveoli and is then diluted in some 2000 cc. of residual air.

It is possible that this bronchiolar filter might be capable of furnishing both heat and moisture for inspired air in the absence of nasal function. That would depend upon the volume of blood flow and the production of respiratory mucus.

SUMMARY

There is a small progressive increase in the volume of the tracheobronchial tree from the larynx to the bronchi measuring 1.5 mm. in diameter. It is from 50 to 100 per cent. In this portion of the tree there does not seem to be any very effective air cleansing device except the direct impingement of the air on the mucous blanket lining the tree. From fragmentary data available, there seems to be very considerable expansion in the volume of the tract in tubes less than 1.5 mm. in diameter. Before the cilia cease, the tree has divided into bronchioles which number at least somewhere between 15,000 to 70,000, each of which is lined with ciliated epithelium and a sticky mucous blanket. The composite circumference of these tubes measures many meters. This, "the bronchiolar filter," is an effective, final device to protect the alveoli from dust and fumes that might gain access to the trachea. One infers that the cilia move off the mucous blanket as is the case elsewhere in the tract. One might also infer that this bronchiolar filter could be even more effective in the matter of dust removal and humidification, if sufficient secretion is produced, than the nose. It does not, of course, protect the trachea and the larger bronchi from improperly prepared air.

324 West Superior Street.

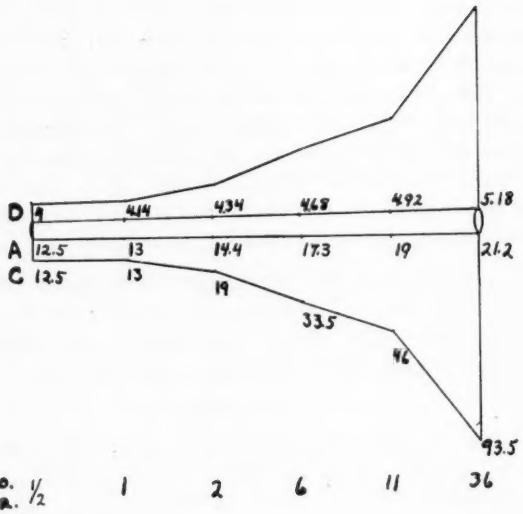

D 9 4.44 4.34 4.68 4.92 5.18
A 12.5 13 14.4 17.3 19 21.2
C 12.5 13 19 33.5 46 93.5
No. Br. ½ 1 2 6 11 36

Fig. 2.—Composite measurements at six successive levels of the bronchial tree from the left lung of a stillborn infant. The bronchial tree was slit posteriorly, and the circumference of the trachea and all bronchi down to an average size of 0.83 mm. in diameter were measured. The comparative composite volumes of the tracheobronchial tree at the six different levels are shown constructed into the form of a single tube. The tracheal end is toward the left. Figures opposite "D" represent the calculated diameters of the composite single tube, and those opposite "A", the summation of the calculated cross-sectional areas of the bronchial tree at the six different levels. There is a rather small increase in total volume, as indicated by the increase in diameter from 4 mm. to 5.18 mm. The figures opposite "C" represent the summation of the measurements of circumferences. The area between the diverging lines represents diagrammatically the actual area of the mucous membrane lining the tracheobronchial tree. The figures below (*no.Br.*) indicate the number of bronchi measured at each level. The reason that the trachea is given as ½ is that only the left lung was measured. It is assumed that half the air passing through the trachea goes to the left lung. The calculated diameter of a tube of half the volume of the trachea measured in this instance would be 4 mm. The actual diameter of the trachea was 5.76 mm. (calculated from the measurement of the circumference).

D—Calculated diameters of the composite single tube having the same volume at the various levels as the bronchial tree. *A*—Summations of the calculated cross-sectional areas of the bronchial tree at the various levels. *C*—Summations of all measured circumferences at the six various levels.

No.Br.—Number of bronchi measured.

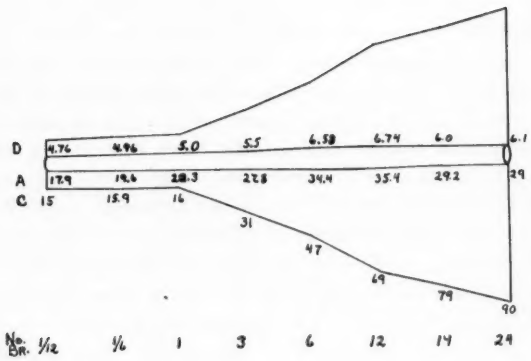
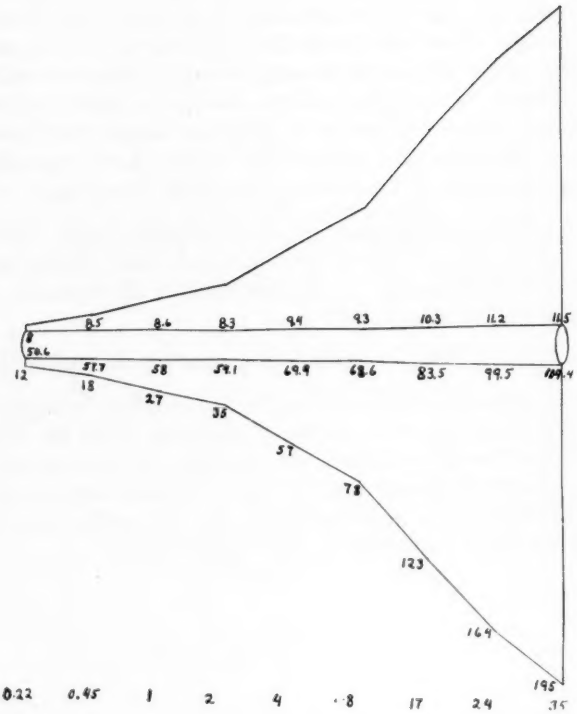

D
A
C
4.76
4.96
5.0
5.5
6.58
6.74
6.0
6.1
17.9
19.6
28.3
27.8
34.4
35.4
29.2
29
15
15.9
16
31
47
69
79
90
No. BR.
1/12
1/6
1
3
6
12
19
29

Fig. 3.—Composite measurements at eight successive levels of the bronchial tree of the middle lobe of the right lung from a 44-year-old man. From the measurements obtained, it was estimated that 1/6th of the air passing through the right bronchus and 1/12th of that passing through the trachea entered the right middle lobe. The diagram shows the size of a composite single tube having the volume of the bronchial tree at the various levels, including the estimated fractions of the trachea and right bronchus.

D—Calculated diameters of the composite single tube. *A*—Summation of the calculated cross-sectional areas of the bronchial tree at the various levels. *C*—Summations of all measured circumferences at the various levels.

No.Br.—Number of bronchi measured at the various levels.

As in the case of the infant (Fig. 2), there is a comparatively small increase in the total volume of the tract from the trachea to the smallest bronchi measured. In this case, the average size of the smallest bronchi measured was 1.2 mm. The total area of the lining mucous membrane, as represented by the diverging lines, shows a marked progressive increase at the various levels.


8.5
8.6
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9.3
10.3
11.2
8
58.6
57.7
58
59.1
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68.6
83.5
99.5
12
18
27
38
57
78
123
164
195
0.22
0.45
1
2
4
8
17
24

Fig. 4.—Composite measurements at nine successive levels of the bronchial tree of the lower lobe of the right lung from a man 44 years old. From the measurements obtained, it was estimated that 22 per cent of the air passing through the trachea and 45 per cent of that passing through the right bronchus entered the right lower lobe. The size of the composite tube shown is calculated at the first two levels on the basis of these percentages. The diagram of the composite tube indicates the total volume at the various levels. Again, as in the case of the middle lobe and of the left lung of the infant, there is a small progressive increase in volume at the successive lower levels as indicated by the increase in diameter from 8 to 10.5 mm. The average diameter of the smaller bronchi measured in this specimen was 1.76 mm.

D—Calculated diameters of the composite single tube of the same volume as the bronchial tree. *A*—Summations of the calculated cross-sectional areas of the bronchial tree at the various levels. *C*—Summations of all measured circumferences at the nine various levels.

No.Br.—Number of bronchi measured.

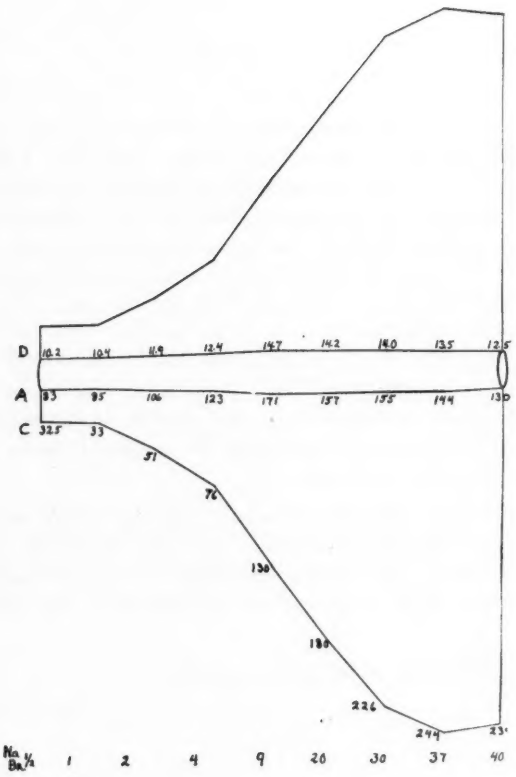
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Fig. 5.—Composite measurements at nine successive levels of the bronchial tree of the left lower lobe from a man 83 years old. The left hand figures represent the left bronchus. No measurement of the trachea was obtained in this case. There was considerable broncho-pneumonia in this lung and many of the membranous bronchi were dilated and full of purulent secretion. This may account for the increased volume in the middle of the composite tube shown in the diagram as compared with the two ends. It may also account for the greater composite circumference at the eighth level as compared with the ninth. On the other hand, it may be that some bronchi at the ninth level were missed. The diameter of the composite tube in this case increased from 10.2 mm at the right bronchus to 12.5 mm. at the level of the smallest bronchi measured (in this instance, 1.74 mm.).

D—Calculated diameters of the composite single tube having the same volume at the various levels as the bronchial tree. *A*—Summations of the calculated cross-sectional areas of the bronchial tree at the various levels. *C*—Summations of all measured circumferences at the nine various levels.

No.Br.—Number of bronchi measured.

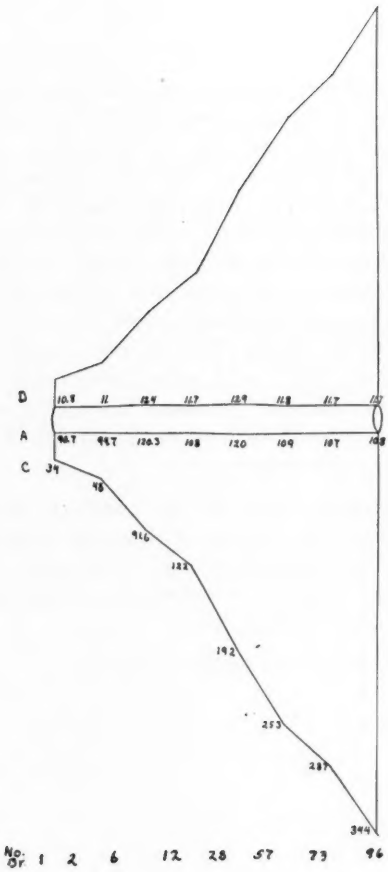
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Fig. 6.—Composite measurements at eight successive levels of the bronchial tree of the left lower lobe from a man 34 years of age. The left hand figures represent the left lower lobe bronchus.

The progressive increase in volume was quite small in this case, as indicated by the increase in cross-sectional area from 90.7 to 108 sq. mm. The increase in the area of the mucous membrane, on the other hand, was very striking. The total of the added circumferences increased tenfold between the first and the eighth levels. The specimen was a good one and it was possible to measure the bronchi down to an average diameter of 1.12 mm.

D—Calculated diameters of the composite single tube having the same volume at the various levels as the bronchial tree. *A*—Summations of the calculated cross-sectional areas of the bronchial tree at the various levels. *C*—Summations of all measured circumferences at the eighth successive levels.

No.Br.—Number of bronchi measured.

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XXX

PROBLEMS OF DIAGNOSIS IN OBSTRUCTION OF THE EUSTACHIAN TUBE

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WATERVILLE, MAINE

While the diagnosis of obstruction of the eustachian tube is generally quite obvious, the determination of the underlying or predisposing cause may, at times, prevent real difficulties.

This paper will be concerned largely with a discussion of causative factors, although some consideration of symptoms and treatment seems unavoidable. More or less emphasis will be placed upon the nasopharynx, as neoplasms in this area are frequently overlooked. Four case reports, illustrative of certain diagnostic pitfalls, are included.

In the earlier days of otology a great deal of emphasis was placed upon so-called "catarrhal deafness" and inflation was the popular method of treatment. In 1921 Emerson[1] called attention to the relatively greater importance of perception deafness, which he considered as usually due to focal infection, and soon the eustachian tube lost caste, so to speak.

The last 20-odd years produced a much better understanding of the physiology of hearing and the pathology of deafness, due to brilliant researches, such as the work of Wever and Bray, Lorente de No, Davis, Lurie, Hughson, Crowe and Guild. The tremendous developments in aviation and the recognition of aerotitis as a clinical entity reawakened interest in the eustachian tube and in the importance of maintaining its normal function. Wolff's[2] histological studies have provided a better understanding of the anatomy of the tube. An excellent description of the anatomy, physiology and histopathology is found in Eggston's and Wolff's[3] recent textbook. All this, together with more accurate methods of testing hearing, should eliminate the vague and meaningless diagnosis of "catarrhal deafness" and allow a better determination of those cases in which tubal obstruction is a factor. Undoubtedly the initial

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From the Thayer Hospital.

symptoms and predominant signs in many patients with impaired hearing, especially in the younger age group, are due to tubal obstruction. The mistake, in the past, was not only in attributing far too many cases of hearing impairment to this condition but too often accepting this symptom as a clinical entity, failing to recognize the underlying causes.

More often than not, the patient volunteers the statement that the ear feels "full" or "blocked," and that his own voice sounds loud and resonant (autophony). Excepting for cerumen blocking the external meatus, the commonest cause of these symptoms is an inflammatory reaction blocking the eustachian tube. Hearing is impaired and there is usually a varying degree of tinnitus. The condition may tend to subside within a few weeks, sometimes to recur intermittently, depending upon the underlying cause. In certain cases of infectious origin this may be the forerunner of acute otitis media. Should the obstruction persist, developing chronicity, the patient may become adjusted to this feeling of fullness and complain mainly of deafness and tinnitus. In young children this complaint is not so commonly elicited, probably because the obstruction is more constant and more easily accepted.

The examination usually reveals retraction of the membrana tympani, although at times it may appear quite normal. The eustachian tube does not open readily to usual methods of examination and sometimes evidence of secretion may be heard in the auscultation tube on attempting to catheterize. Sometimes a fluid level, indicative of hydrops, may be noted. Crowe[4] has shown that the audiogram may exhibit a greater drop in the upper tonal range. This may be even more noticeable with the monochord. Hearing by bone conduction is generally unaffected. The Rinne test is negative, and the Weber test lateralized to the affected side in unilateral cases. All this may be confused if superimposed upon an already existing hearing defect of the perception type.

Inflation by politzerization or by catheterization may relieve the symptoms. Often the best means of affording relief, especially if there is any evidence of secretion in the middle ear, is by paracentesis, followed by careful aspiration. However, treatment, if it be more than palliative, should be directed toward the underlying or predisposing cause. There is still a perhaps unconscious tendency to think of this condition as a clinical entity, with the danger that we may be making use of a diagnostic wastebasket. It would seem advisable, always, to consider tubal obstruction as resulting from any condition capable of altering the normal physiological action

of the eustachian tube. It is axiomatic that intelligent therapy is dependent upon correct diagnosis.

In children nasopharyngeal lymphoid hyperplasia is a common cause and is not an absolute rarity in adults, although frequently overlooked. Eggston and Wolff[3] state that Rosenmüller's fossa is "normally packed with small well-organized lymph nodules" but that they did not find pathological invasion of the lumen of the tube in their microscopic sections. However, lymphoid tissue in the lower third of the tube has been reported by the Hopkins group and by Mosher.[5] Careful adenoidectomy, performed under direct visual control, is generally effective. This should be followed by postoperative inspection of the nasopharynx and audiograms; and whenever indicated, by irradiation, using the Crowe technique.

While our experience with irradiation is somewhat limited, we have had better results in children and when it was used as an adjunct to surgery. While irradiation alone has given temporary relief of symptoms in three cases in adults treated within the year, this has been followed by recurrences later. I now prefer to remove even small amounts of adenoid tissue and to follow with irradiation for its effect upon the tubal orifice. This seems the more logical procedure.

Tubal obstruction not infrequently follows some acute infection of the upper respiratory tract, especially if the nose has been blown violently. Postnasal discharge from a sinusitis, crusting from atrophic rhinitis, or altered air currents from intranasal abnormalities, all may interfere with tubal patency. Hoople and Blaisdell[6] found the majority of their cases due to infection of the nose or throat, about 12 per cent of allergic or endocrine origin, and a scattering following pregnancy, aerotitis, or trauma.

At times dental malocclusion may result in compression of the tube with symptoms of obstruction, which will only be relieved by correction of the bite.

Oftentimes the solution of the problem may be found in the nasopharynx if a thorough, painstaking examination be carried out, anesthetizing the pharynx, if necessary, and making use of the nasopharyngoscope and direct inspection by elevation of the soft palate, as well as using the mirror. Fullness in the ear and tinnitus may be the outstanding symptoms of residual adenoid tissue, nasopharyngeal cyst, either embryonal or retention, a hypertrophied posterior tip of the inferior turbinate, or hypertrophic lateral pharyngeal bands. Adhesions across the fossa of Rosenmüller are seen so fre-

quently in patients with absolutely no symptoms that it seems doubtful if they themselves predispose towards tubal obstruction, unless they harbor infected lymphoid tissues deep in the fossa. Occasionally we may see adhesions across the orifice or scarring from a poorly performed adenoidectomy.

The possibility of nasopharyngeal neoplasm causing tubal obstruction always should be kept in mind, especially in unilateral cases. Careful inspection of the nasopharynx may show suspicious tissue, either in the vault or the fossa of Rosenmüller and biopsy establish the diagnosis. Lateral roentgenograms may give useful information. Cervical adenopathy has great diagnostic significance, the importance of which cannot be overstressed. Too many cases, unfortunately, are not seen or not diagnosed until there is this visible evidence of lymphatic extension which renders the prognosis very poor. While many of these nasopharyngeal neoplasms are quite radiosensitive and respond rather dramatically to therapy, the treatment of metastases is generally futile. Only early recognition of the primary lesion offers hope of a cure.

REPORT OF CASES

Sometimes the diagnosis of neoplasm may be missed because of the presence of symptoms of other conditions more commonly considered as predisposing causes.

CASE 1.—Mrs. McC., age 49, was previously seen by two very well-trained and competent otologists because of a complaint of fullness and obstruction in the right ear. The left ear had been deaf for 20 years (Fig. 1). She had nasal obstruction and a mucoid discharge. The condition was attributed to allergy and treated as such.

She was referred to me one month later, at which time she had a typical hydrops of the middle ear. The examination showed a suspicious mass in the nasopharynx and an enlarged cervical node on the affected side. The biopsy was reported as "lymphoma." Intensive roentgen therapy gave only temporary relief, death occurring four months later.

Sometimes repeated biopsies are necessary before one can establish the diagnosis. The following case illustrates the peculiar invasive character of some of these tumors.

CASE 2.—Mr. J. N., age 74, was seen recently because of sudden deafness in the left ear of four months' duration. He had had a chronic sinusitis for years, which had been treated in his home city.

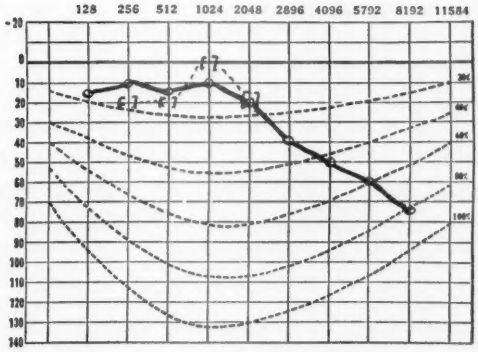

Fig. 1, Case 1.—Hydrops of ear secondary to malignant lymphoma of the nasopharynx. Audiogram of right ear only, made September 3, 1948. Whispered voice 1/20; Rinne test 45/17; Weber test not lateralized.

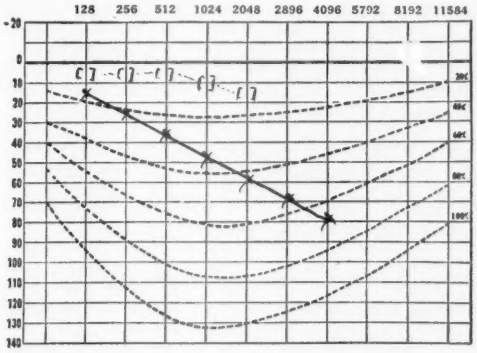

Fig. 2, Case 2.—Obstruction of left eustachian tube secondary to lymphosarcoma involving the orbit. Audiogram of left ear made January 20, 1948. Spoken voice 1/40; Rinne test 20/20; Weber test lateralized to left.

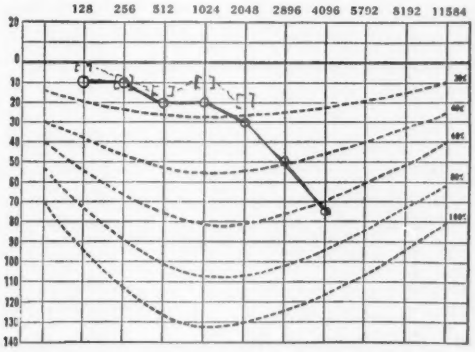

Fig. 3, Case 4.—Hydrops of right ear, secondary to reticulum-cell sarcoma. Audiogram of right ear only, made April 8, 1947. Whispered voice 8/20; Rinne test 50/18; Weber test lateralized to left.

His present illness began eight months before with the development of diplopia in the left eye. Later he noted pain in the temporomandibular joint on opening the jaw. Soon there was a complete loss of vision in the affected eye and numbness, noted at times, in that side of the face. When seen he complained of pain about the orbit.

Ophthalmological examination by Dr. H. F. Hill revealed a marked proptosis, almost complete loss of oculomotor function and an optic atrophy. This indicated some invasive process involving the orbit.

Examination of the ears showed a somewhat retracted membrana tympani on the left. The audiogram showed a drop along the whole tonal range and hearing for conversational voice was reduced to one foot (Fig. 2). The right middle turbinate was missing and there was a small polyp in the posterior ethmoid region.

Examination of the nasopharynx showed a rather firm but polypoid-appearing mass obstructing the orifice of the eustachian tube and originating on the anterior part of the torus. Gently inflating the tube by passing a catheter over this mass and into the orifice relieved the pressure and improved the hearing. There was no evidence of cervical adenopathy and the remainder of his examination was essentially negative.

X-ray examination showed the sinuses on that side hazy. Lipiodol was injected into the antrum and showed normal filling.

Repeated biopsies of specimens from the tubal orifice and the ethmoid region were reported as "benign polyp, marked edema, chronic inflammation." Finally as we were preparing to explore the orbit, a specimen taken from deep in the fossa of Rosenmüller was reported as "lymphosarcoma." Roentgen therapy has resulted in marked improvement with recession of the proptosis, restoration of oculomotor function and improvement in hearing.

It is interesting to speculate as to the origin of this tumor. While a nasopharyngeal origin would be the more common explanation, from the chronological sequence of symptoms it might seem that it originated in the orbit, as diplopia was the first one noted. Eggston and Wolff[7] report finding lymphoid neoplasms in the orbit. They also state that lymph follicles are frequently present in chronic infection of the sinuses. Possibly the orbit was invaded from the ethmoid which was known to be the site of chronic infection. Extension posteriorly could explain the tubal obstruction.

Distortion of the torus may suggest the presence of a neoplasm but may be of luetic origin. This is illustrated by the following case.

CASE 3.—Mr. F. P., age 57, had had a bilateral hearing impairment of many years' duration. Two weeks before being seen the right ear suddenly became "blocked" and the hearing much worse. He had a luetic history but was reported to have had adequate treatment and the blood serology was negative. There was a perforation of his nasal septum and a typical old luetic scar on the soft palate. Nasopharyngoscopy revealed a distorted torus, the posterior lip being smooth, rounded and almost obliterating the tubal orifice. The tube was not patent to catheterization. There was clinical evidence of a chronic sinusitis and roentgenograms showed a markedly involved antrum on the same side.

Despite the negative serology, a luetic lesion involving the torus, complicated by the sinusitis, seemed the most likely diagnosis, although the possibility of neoplasm was kept in mind. An operation on the antrum revealed a markedly hyperplastic lining membrane with multiple abscesses. A subsequent blood Hinton test was reported positive and he was placed upon antiluetic therapy. His condition rapidly improved and the complaint of fullness disappeared.

He has been under observation for the past five years. There has been no further loss of hearing and while the torus is still distorted, the tube remains patent. It seems probable that a luetic lesion of the torus was aggravated by discharge from the infected antrum resulting in tubal obstruction.

At times it may be exceedingly difficult to ascertain the underlying cause of obstructive symptoms and only the subsequent development of visual evidence may disclose the true pathological condition.

CASE 4.—Dr. C. C., age 70, was referred with a history of the right ear suddenly becoming full and obstructed. He had had a perception deafness in the left ear for many years (Fig. 3). Examination showed a typical hydrops of the right middle ear. Careful inspection of the nasopharynx was entirely negative. Paracentesis, followed by inflations, gave temporary relief.

He was not seen for two months, when nodes were noted at the angle of the jaw and over the parotid gland. At this time the ex-

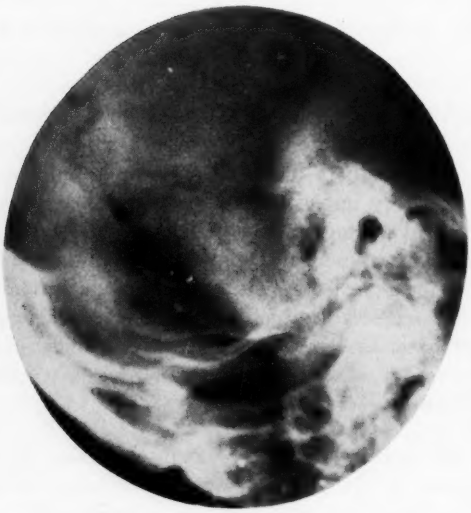

Fig. 4, Case 4.—Roentgenogram of mastoid showing defect due to reticulum-cell sarcoma.

ternal auditory meatus was found almost occluded by a soft, bluish, nontender mass situated in the anterior-inferior wall. The nasopharynx still appeared normal. The biopsy of a node was reported as "malignant lymphoma, possibly reticulum cell sarcoma."

Roentgenograms of the mastoid showed a large defect, somewhat similar to that seen after mastoidectomy (Fig. 4). Chest films were normal except for some enlarged hilar glands. Roentgen therapy resulted in complete regression of the tumor and re-establishment of tubal patency.

In this case the obstruction simulated the tubal type when first seen. Earlier x-ray examination would have been advantageous. It is probable that this tumor originated in lymphoid tissue in the eustachian tube itself but too deep to be seen on nasopharyngoscopy. While apparently controlled at present, the time elapsed, one year, is too short for one to be sanguine regarding the prognosis, although localized reticulum-cell sarcoma responds well to adequate radiation. Craver[8] states that the possibility of curability of early lymphoid lesions of this type depends primarily on whether the process is of unicentric origin.

SUMMARY

Symptoms due to tubal obstruction are fairly common complaints among patients seeking otological consultation. The commonest causes are infections of the upper respiratory tract and lymphoid hyperplasia in the nasopharynx. The diagnosis of these, as well as of aerotitis, is usually obvious. Allergic and endocrine conditions, as well as dental malocclusion may be factors in producing tubal obstruction. Many times the underlying cause may be found in the nasopharynx. Residual adenoid tissue, hypertrophic lateral bands, cysts, hypertrophic posterior tips of inferior turbinates, or neoplasms may cause obstruction. Nasopharyngeal tumors may be overshadowed by more common conditions or be unrecognized until the development of cervical adenopathy. Too much reliance should not be placed upon one or two negative biopsies when the clinical picture is consistent with neoplastic disease.

CONCLUSIONS

Obstruction of the eustachian tube, whether acute or chronic, should not be considered as a clinical entity. It may result from any deviation from the normal capable of altering the physiological action of the tube. Treatment, other than palliation, should be directed towards the underlying cause. Careful study of the nasopharynx is always indicated but often neglected. Cervical adenopathy has great significance.

The writer desires to express his appreciation for the helpful criticism of Dr. Harris P. Mosher in preparing this paper.

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XXXI

TONSILLECTOMY IN THE UNITED STATES

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MINNEAPOLIS, MINN.

This should be a worn-out subject. It is the eighty-third paper on some aspect of the tonsil problem to be presented before the American Laryngological Association in its 69 meetings. However, there is evidence that a problem concerning the tonsil still exists. In the five years 1942 to 1946 inclusive, there has been an average of over one hundred papers per year concerning tonsils listed in the *Quarterly Cumulative Index,* and this in spite of the war years when medical literature was less voluminous than in ordinary times. In addition, lay publications have aired and emphasized the negative side of the problem. The *Woman's Home Companion,* in July, 1947, publicized the tonsil business under the title "Unnecessary Operations." A few months later the *Reader's Digest* summarized this article for wider lay consumption. The following is quoted from that summary:

> "More tonsils are yanked out of the American anatomy than any other organ. The well-known pediatrician, Dr. Harry Bakwin, is the latest to lash out at the unwise but widespread practice of removing tonsils indiscriminately. He points out that while tonsillectomy is considered a relatively safe minor operation, it sometimes results in death and oftener in serious complications such as pneumonia or lung abscess.
>
> "The absurdity of indiscriminate tonsil operations was demonstrated some years ago in a study of a thousand eleven-year-old children conducted by the American Child Health Association. Three-fifths already had their tonsils removed. The other four hundred were examined by a group of doctors who decided that half of them needed tonsil operations. Those who passed were examined by a second group of doctors. Again, half were chosen for tonsil operations. After the process was repeated for the third time only sixty-five children were left with their tonsils intact. Some of these no doubt would have been marked for an operation if a fourth doctor had been called in.
>
> "The directors of this classic survey concluded that tonsil operations depended not so much on the child's health as on the vagaries of a particular doctor."

My purpose in this presentation is to raise a dissenting voice to the contention that the tonsil and its surgical treatment need be a problem. When a tonsil operation is performed by one adequately trained and with good technique, it is a highly satisfactory procedure in terms of the end results. At this point I would state, however, that in a majority of cases, I have not found it to be a simple

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Read at the Sixty-Ninth Annual Meeting of the American Laryngological Association, Hot Springs, Va., April 14, 1948.

minor surgical procedure, but that it is not followed by any serious morbidity and should never result in a mortality.

In the writings, both medical and lay, on this subject, there seem to be three aspects of tonsillectomy which constitute the problem part of the matter. These are: (1) the indications for tonsillectomy, (2) the adequacy of the operation and (3) the morbidity and even the mortality connected with tonsil surgery.

The Indications for Tonsillectomy.—I have personally performed tonsillectomy on slightly more than 3,000 persons. Practically all of the children on whom I operate are referred by pediatricians who are acquainted with the whole picture of the particular child's health. Most of my adult patients are under the care of an internist, or are persons in whom there is no doubt about their needs because of very obvious local disease or because they have repeated attacks of tonsillitis. Out of this experience I have simplified the indications into three categories, as follows:

1. Repeated attacks of tonsillitis.
2. Hypertrophy to the extent of obstruction.
3. Evidence of persistent chronic infection.

There should be little argument concerning the first and second indications. Simple tonsillar hypertrophy is common in early childhood and in itself is not a need for tonsillectomy. When the hypertrophy is excessive, the child is invariably a mouth breather even though the adenoid may not be obstructive. Also, he eats poorly. In a majority of these instances, a considerable weight gain is experienced after the tonsils and adenoids are removed.

The third category is one in which there is often the element of chance that tonsillectomy will not relieve a condition in which a focus of infection is thought to be a factor in the morbidity. However, the chance seems worth taking.

There are many aspects of this third category concerning which the pros and cons could be discussed. Not infrequently cases are encountered in which the family or the doctor are interested in tonsillectomy to prevent head colds. When the tonsils and adenoids are obstructive or obviously diseased, the benefits from tonsillectomy and adenoidectomy in the prevention of colds not of virus origin are in most cases very obvious. When the tonsils and adenoids are within normal limits and removal is advised as a prophylactic measure, the end results are often disappointing. Some physicians have ex-

pressed a belief that removal of tonsils and adenoids have rendered a child more susceptible to head colds. It is possible that a protective function of the tonsils and adenoids does play a role in this respect. However, the fact is often overlooked that the early school age is usually the child's most susceptible period in the matter of respiratory infections, a period in which he may be developing some immunity, and that the removal of his tonsils and adenoids in a preschool period may have little to do with the relatively increased frequency of upper respiratory tract infections after he is exposed to the infections so prevalent in the early years of school life.

The Adequacy of Tonsillectomy.—Much has been written about incomplete tonsillectomy and the importance of tonsil remnants which are often referred to as "stumps" or "tags."

Many of you will remember an editorial in the *Journal of the American Medical Association*[1] in 1928 that had apparently been inspired by two contemporary reports, one[2] from data which indicated that one-third of all operations performed since 1924 on American urban population was done for the removal of tonsils and adenoids, and the other the studies of Rhoads and Dick[3] describing the high incidence of postoperative tonsillar remains and the potent bacterial content of these remnants. Rhoads and Dick had found fairly large pieces of tonsillar tissue remaining in the throats of 290 of 403 nurses (73 per cent) entering training at the Presbyterian and Cook County Hospitals in Chicago. When some of these pieces of tonsil tissue were removed and their bacterial content studied quantitatively, it was found that a larger count of pathogenic bacteria could be found in a gram of tonsillar remnants than in an equal amount of unoperated tonsillar tissue.

Now, nearly 19 years later in the same *Journal of the American Medical Association* for June 21, 1947, there appears under "Current Comment"[4] the following statement in part:

> "Tonsillectomy is the most frequent surgical procedure in the United States . . . The results of the analysis of the rate of recurrences of tonsillar growth after tonsillectomy indicate that tonsillectomy should command all the respect of a major operation. The incomplete removal of tonsillar tissue and the subsequent formation of infected tonsil stumps are rather common. The Spuths recently reported the presence of tonsil stumps in 80 per cent of 285 tonsillectomized patients. Instead of being relieved through tonsillectomy of their foci of infection, many patients continue to absorb bacteria and toxins from the tonsillar tissue left in the throat and are often systemically much worse off than prior to their operation. While there is drainage of infections and toxic material from the external surface of the tonsils before tonsillectomy so that only a part of such harmful matter is absorbed into the organism, this route of escape becomes obliterated after tonsillectomy, for the infectious and toxic material contained in the usual tonsil stumps are covered by scar tissue. It is resorbed in its entirety through

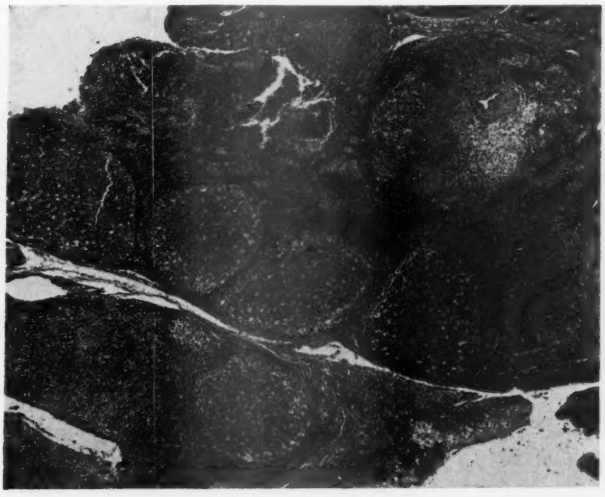

Fig. 1.—Low power magnification of a portion of a plica triangularis showing numerous lymph follicles. The plica was thoroughly removed after the tonsil had been enucleated with a guillotine.

the lymphatics and veins and may cause, in addition to fatigue, heart palpitations, vague muscular pain, secondary infectious and allergic complications in remote organs (polyarthritis, nephritis, endocarditis, asthma, chronic phlebitis). Sometimes the diagnosis of tonsil stumps cannot be made by an inspection of the tonsillar region because the retained tonsil tissue may be buried beneath a thick layer of scar tissue. However, as inflamed tissues, the presence of which is usual with tonsil stumps, are sensitive to pressure, tonsil stumps can be detected with reliability by exerting pressure either directly on the tonsillar scar tissue or externally from the angle of the lower jaw in the direction of the tonsillar fossa. Tonsils when functioning normally are protective organs and should not be removed. Definitely infected tonsils and especially tonsil stumps should be radically removed under general anesthesia and with adequate surgical technic and skill."

This high incidence of pieces of tonsillar tissue remaining in tonsillar fossae (in three-fourths of the cases) seems almost unbelievable. It is probable that in some instances the observer confused simple lymphoid nodules with an actual tonsil remnant.

It has been my observation that in a majority of instances in which there has been complete removal of the faucial tonsil with the capsule intact and in which the plicae are thoroughly removed, small lymphoid nodules grow into the fossae from the membrane lining and fossae. This is particularly true in children. The follicles are

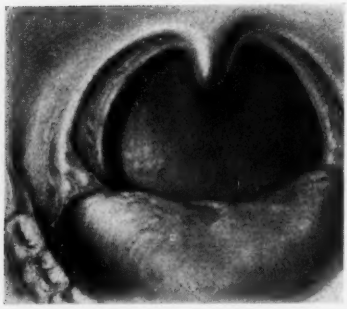

Fig. 2.—The usual picture of a remaining plica triangularis in the right fossa. This is a drawing of an adult whose tonsils were removed in early youth. The presence of this plica had caused no morbidity.

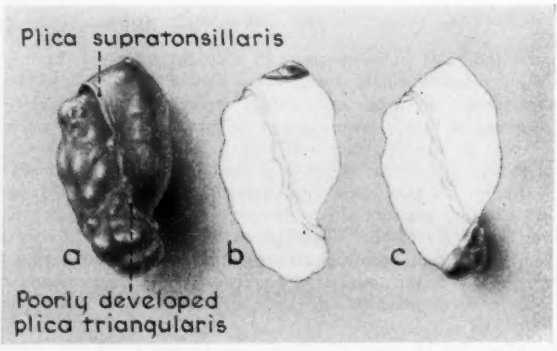

Fig. 3.—(*a*) A large soft tonsil cleanly and completely enucleated. The plica supratonsillaris is intact. In this type of tonsil development, the plica triangularis is usually poorly developed. Occasionally guillotine enucleation misses a piece of tonsil at the upper pole (*b*) or cuts across a portion of the lower pole (*c*). The tonsil should be carefully inspected as soon as it has been enucleated in a search for a defect of this type.

no different from the lymphoid nodules seen on the posterior pharyngeal wall. The younger the patient, the greater the tendency for these nodules to appear. Also, they appear in greater abundance in the patient who suffers from an allergic rhinitis.

Lymphoid structures with germinal centers can be found throughout the respiratory tract and in the digestive tract in Peyer's patches and in the appendix. Presumably, their role is a protective one when they are not diseased. There is some recent evidence that lymphoid tissue is the site of antibody formation.[5]

A tonsil cleanly enucleated cannot grow back. As part of this clean enucleation it is advisable to remove thoroughly the plicae and the prominent, immediately adjacent projection of the lingual tonsil. If the well-developed plica triangularis is left, the lymphoid follicles often present in it (Fig. 1) tend to hypertrophy and sometimes fill the lower portion of the fossa. This is more likely to occur in small children. In my opinion the hypertrophied lymphoid tissue of a remaining plica triangularis (Fig. 2) does not have the pathologic potentialities found in the remnants of the main tonsil mass with its crypts which branch.

Fig. 4.—A large soft tonsil cleanly enucleated with the guillotine. The enlargement (indicated by the arrow) which bulges the capsule may be cut across by the guillotine.

Leshin and Pearlman[6] in a study of the incidence of these follicles found them to be present in 66.6 per cent of sections from the plica triangularis, in 30 per cent of the supratonsillar and in 47.7 per cent of the retrotonsillar plica.

A majority of otolaryngologists use some form of guillotine for tonsil operations on children. I prefer it for the large, soft tonsils of children but agree with Kelly[7] in the preference of a dissection technique for the recessed or submerged tonsils which may be more or less adherent. Whenever a guillotine is used, it is important to inspect the capsule and the plical attachments as soon as the tonsil has been enucleated. One occasionally finds one of the following defects in what he assumes is a complete enucleation:

A portion of the plica supratonsillaris is missing.

There is a defect at the upper pole with a small piece of tonsil tissue missing. It can be found by a careful inspection of the fossa at the site of the defect (Fig. 3b).

The lower pole has been cut across (Fig. 3c).

A projecting enlargement bulging the capsule such as is often seen in the large, soft tonsils of children is sometimes cut across by the guillotine and a piece is left in the fossa (Fig. 4).

Figure 5 depicts the completely healed fossae with hypertrophy of the remnants in instances of tonsil pieces left (*a*) at the left upper pole and (*b*) at the base in tonsillectomy by a guillotine.

Because the two surveys referred to in the *Journal of the American Medical Association* indicated that tonsil remnants existed in approximately three-fourths of the individuals examined, I made a personal study of a group of patients of all ages passing consecutively under my observation. A critical inspection was made of the fossae of tonsillectomized patients to determine in what percentage tonsil remnants existed. Also, I determined in which cases the tonsillectomy had been done by specialists and in which the general practitioner had performed the operation. When the latter fact could not be reliably determined the case was not included in this series. Four hundred and thirty tonsillectomized patients were studied. The following data was obtained:

NUMBER OF TONSILLECTOMIES		PER CENT	TONSIL REMNANTS	PER CENT	LYMPHOID INGROWTHS	PER CENT
Performed by specialists	164	38.1	14	8.5	41	25
Performed by general practitioners	266	61.9	121	45.5	76	28.5
Total	430	100.0	135	31.4	117	27.2

The figures which I have determined from personal observation do not compare with those of Rhoads and Dick or the Spuths. In my series, there were actual tonsil remnants in 135 of the 430 cases, or in 31.4 per cent as against the 73 to 80 per cent of their series. I have suggested that in some instances the observer may have confused simple lymphoid follicles with an actual tonsil remnant. But even if I add the instances of lymphoid ingrowths to the cases of actual tonsil remnants and call all of these tonsil "stumps" or "tags," I get only 58.6 per cent.

Campbell[8] in 1939 reported observations on 621 cases of tonsillectomized young women between 18 and 21 years of age. When what he considered to be tonsil remnants were found they were classified as infected or noninfected. If the remnants appeared to be simply noninflamed lymphoid tissue, without observable crypts containing exudate or without signs of being partially obscured by scar

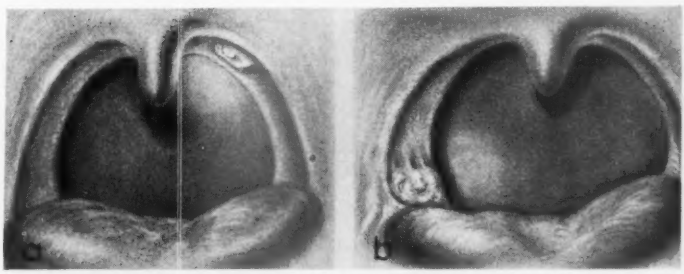

Fig. 5.—Completely healed fossae, with hypertrophy of the remnants, in instances of tonsil pieces left (*a*) at the left upper pole and (*b*) at the base in guillotine tonsillectomy.

tissue, they were classed a noninfected. If the remnants showed signs of chronic inflammation or if there were crypts containing exudative material or if they were imbedded and partially covered by fibrous scar tissue, they were classified as infected. Campbell reported that 77.3 per cent of the 621 students showed tonsillar remnants in the fossae. Of these, 66.6 per cent were placed in the class of noninfected remnants and 10.6 per cent in the class of infected remnants. I suspect that his "noninfected" remnants correspond to what I consider to be lymphoid ingrowths, which in my opinion are not tonsil remnants. In Campbell's series, data on only 153 of his cases were obtained as to whether the tonsillectomy had been performed by one specially trained or by the general practitioner. He found 74.7 per cent of the patients who were operated on by specialists showed tonsil remnants but did not give any figures on this 74.7 per cent as to how many of the remnants were of the noninfected or simple lymphoid type. If I apply his percentage of 66.6 per cent of noninfected remnants of his total of 621 to the 153 tonsillectomies done by specialists, which he studied, the percentage of infected remnants, "tags" or "stumps" approximates 8 per cent and corresponds to the percentage of remnants left by specialists in my series.

The report of the Spuths,[9] which was the basis for the comment in the *Journal of the American Medical Association* in July 1947, stated that three-fourths of a series of 285 new patients who had had tonsillectomies exhibited infected tonsil stumps and lingual tonsils. They claim that: "The majority of people with infected tonsil stumps have very few complaints referable to their throats. The infection seems to affect the entire body. They usually com-

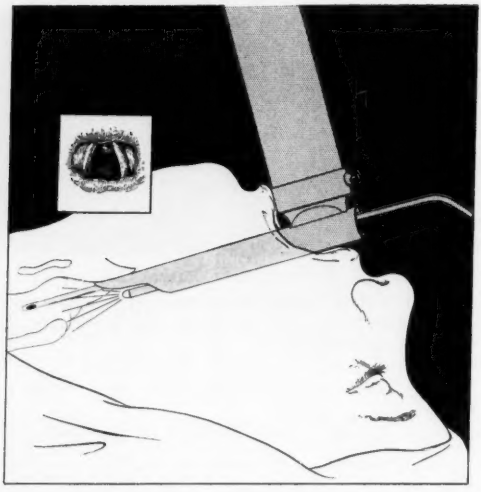

Fig. 6.—Aspiration of the trachea with a laryngeal suction tube through the Flagg laryngoscope is routinely performed in all cases of tonsillectomy done under general anesthesia.

plain of feeling tired, loss of weight, joint pains, shooting pains down the arms and legs, and frequently, bouts of fever. Others may have skin affections and eczemas. In some patients chronic sinusitis or chronic middle ear disease is noted."

They apparently subscribe to the claims of Otto Meyer,[10] who finds tenderness in the submaxillary region, over the tonsil stump and along the course of the jugular vein significant of tonsil stump infection. These writers speak of the removal of the tonsil stump, including *the whole lingual tonsil,* under general anesthesia. I wonder if those who recommend removing the lingual tonsil are convinced that the actual whole of the lingual tonsil is removed. This would require removal of a segment of each side of the tongue extending to the median glosso-epiglottic fold in the midline. In my experience, this is a procedure which from practical considerations should be avoided.

I have asked Dr. C. B. Sputh, Jr., for a statement as to how much of the lingual tonsil they actually remove. He replied, "By careful dissection, we carry a line of cleavage underneath the lingual tonsil from the faucial tonsil. This is then snared off, taking care

that we do not remove any of the musculature or mucous membrane of the tongue."

I believe that this describes the removal of a portion of the lingual tonsil and is comparable to the amount I remove in instances in which there is a hypertrophied prolongation of the lingual tonsil.

In my experience the ordinary lingual tonsil is not a common cause of morbidity and I only occasionally encounter an infected tonsil stump accounting for symptoms and signs such as the Spuths or Meyer describe.

Morbidity and Mortality from Tonsillectomy.—The bugbear of tonsillectomy is postoperative hemorrhage. It is my observation that this is practically needless if an extra few minutes are given to careful hemostasis at the time of tonsil removal. This hemostasis should be performed according to the best standards of surgical technique using tie and suture with a minimum of tissue inclusion or of tissue trauma. This will avoid all but an extremely small incidence of postoperative hemorrhage. Bleeding several days postoperatively, particularly at the end of the first postoperative week, occurs in a small percentage of cases as the membrane lining the fossa separates. As a rule, this offers no great difficulties.

The instances of lower respiratory tract disturbances following tonsillectomy are probably dependent largely on the aspiration of blood, tonsillar debris, and possibly fragments of adenoid tissue into the trachea during the operation. I make it a routine practice to do a direct laryngoscopy at the close of every operation and suck out the trachea with a laryngeal suction tip (Fig. 6). One can introduce this easily down to the carina, and if it is gently done no trauma results. Not infrequently I find it useful to aspirate mucus from the trachea as soon as the child is anesthetized and before starting the operation.

When large children or adults are operated on under general anesthesia, an intratracheal tube provides smoother anesthesia, and it is part of the technique to aspirate the trachea with a catheter inserted through the tube.

I have heard anesthesiologists remark that statistics indicate that the tonsil anesthetic is the most hazardous of general anesthetics. That hazard and fatal hemorrhage apparently are the important contributors to the mortality associated with the operation. Recently, within a twelve-month period I have been asked to express an opinion as to the possible fault in five deaths associated with ton-

sillectomy and adenoidectomy. Three of these seem to be anesthetic deaths and two could be blamed on hemorrhage.

A carefully administered general anesthetic by one adequately trained, with the surgical procedure performed by an experienced operator and hemorrhage controlled, is in my experience a safe procedure.

In a discussion of morbidity associated with tonsil and adenoid surgery in this day, one must consider the possible relationship between the surgical wound created by the operation and a greater susceptibility to poliomyelitis, particularly the bulbar form.

The data accumulated to date suggests that there is no causal relationship. However, to be convincing and according to the proper factors in statistical analysis it seems necessary to introduce the factors of age and the number of tonsil and adenoid operations performed in the area being studied. It is my personal belief that when the facts are all in we will not regard the tonsil and adenoid operation as predisposing to poliomyelitis. However, regardless of whatever is eventually proven regarding this relationship, there should be no more basis for performing tonsil and adenoid surgery during an epidemic of poliomyelitis than there would be during an epidemic of respiratory infection or any other type of infectious disease.

SUMMARY

In the minds of the laity and for some of the medical profession, tonsil surgery is a cause for concern on three counts: (1) the indications, (2) the probable inadequacy of the operation and (3) the associated morbidity and even mortality.

In the hands of the trained laryngologist, none of these three aspects of the tonsil operation need be a cause for concern.

The faucial tonsil which is cleanly enucleated with complete removal of the plicae does not grow back. Lymphoid ingrowths from the lining of the fossa are common. These are often erroneously called tonsil remnants. Ordinarily, these lymphoid ingrowths are not a source of any significant pathologic condition.

MEDICAL ARTS BUILDING.

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XXXII

THE ETIOLOGY, TREATMENT AND PREVENTION OF CHRONIC SINUS INFECTION

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AND

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The ultimate object of all researches on the common cold has been to find a drug, a vaccine or some means to protect against repeated infection with the common cold virus. All such studies so far have been a failure.[1] It has been proven that the common cold is due to a virus, which is an anaerobe that grows and produces clinical symptoms only after it has entered a living cell; also, that the virus must be in contact with a cell for at least 30 minutes before it can gain entrance to the cell and begin to multiply and produce clinical symptoms. This suggests that the common cold virus, which is an intracellar parasite, does not possess all the enzyme systems essential for growth and reproduction, but is dependent on the host cells for these necessary enzymes. It also offers a possible explanation for the fact that virus diseases do not respond like bacterial infections to known chemotherapeutic agents and antibiotics. Most bacteria and spirochetes possess all the enzyme systems essential for growth and reproduction, and their destruction by these therapeutic agents is believed to be due to interference with their enzyme systems or metabolic processes.[2]

Pyogenic bacteria are present in the nasopharynx from infancy to old age. Their first appearance in infants soon after birth often is not associated with an inflammatory reaction or any evidence of infection. There is a growing impression that these pyogenic bacteria, the streptococcus, staphylococcus, pneumococcus and Haemophilus influenzae, are in themselves powerless to initiate infection.[3] The carrier rate of these organisms varies from time to time and year to year. When cultures show a widespread distribution of

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pathogenic organisms in a community, acute upper respiratory infections are usually severe, and ear, sinus and lung complications are common. On the other hand, during the summer and early fall, when cultures usually show that the distribution of pathogenic organisms is relatively limited, the majority of acute upper respiratory infections are mild, complications much less frequent and the usual diagnosis is the common cold. Repeated infections with the common cold virus seem to facilitate the distribution of bacteria in the nasopharynx and prepare the soil for widespread outbreaks of respiratory disease in the community.

In a sense the ear, sinus and lung complications of upper respiratory infections are secondary to the chain of events set in motion by the common cold virus. This virus, being airborne, enters the nose, is carried to the nasopharynx by the mucus and cilia of the nasal mucous membrane, finds a lodging place in the crypts and folds of lymphoid tissue, penetrates the lining epithelial cells, breaking down cell membranes and protective barriers and thus providing a portal of entry for the bacteria that are always present in this location. A cellulitis ensues, the number of bacteria increases, the resulting edema causes obstruction in the nasal passages or eustachian tubes that interferes with drainage, and an acute sinusitis or otitis media results. If the outlet of any air-containing cavity becomes occluded by edema or a fibrin plug, the air within that cavity is soon absorbed, and as the air pressure within the cavity becomes less than the atmospheric pressure on the surface of the body, the blood and lymph vessels in the cavity become increasingly engorged and often rupture. This adds to the obstruction of the only outlet through which drainage can occur. When the eustachian tubes are thus occluded, the resulting changes in the middle ear are called aerotitis; when a sinus orifice is occluded an aerosinusitis ensues, and when a terminal bronchus is occluded it is reasonable to assume an aeropneumonitis follows. If bacteria happen to be present in the area thus shut off, an abscess is formed. This is probably the genesis of bronchiectasis. In the ear and sinuses it is possible to establish drainage by surgical measures. In the lung the only way to establish drainage is to visualize the area through a bronchoscope and with the aid of vasoconstrictors applied locally and point suction, to remove the obstructing plug. By employing these mechanical measures to insure drainage early in the course of the infection before extensive cellular destruction and granulations have formed, and at the same time giving adequate doses of sulfonamides or antibiotics, it is possible to prevent chronic ear and sinus infections and it should be possible to prevent chronic suppurative bronchiectasis.

Lymphoid tissue throughout the body, and especially that in the upper air passages, is extremely resistant to infection with the common pyogenic organisms. The chief function of lymphoid tissue is to filter out bacteria and probably to aid in the production of antibodies. It protects the body against bacterial infection but affords no protection against infection with the common cold virus. Histologic examination of infected tonsils and adenoids, removed at operation, rarely shows an abscess in lymphoid tissue, but we always find a localized or widespread destruction of epithelium lining the crypts. The portal of entry for the bacterial infection probably is often prepared by a preceding virus infection, just as a mechanical or chemical injury of skin epithelium makes conditions favorable for infection by the bacteria that happen to be in that locality, and a cellulitis ensues.

The discovery of a vaccine or drug that would protect against virus infections would be ideal, but until that day comes the otolaryngologist must use all the measures at his command to prevent the common cold and its complications. It often happens that marked improvement in the frequency of upper respiratory infections and their complications follows operative removal of tonsils and adenoids, especially in children. In many of these children, however, the relative freedom from colds and ear or sinus infection lasts for only a few months or a year after the operation. In these cases examination usually shows recurrence of lymphoid tissue, which again affords lodging places for the virus. It is not the size, but the location and contour of these lymphoid nodules that is important. Nasopharyngoscopic examination of a large group of patients in whom the operation had been a failure showed nodules of lymphoid tissue in and around the orifice of the eustachian tubes, in the choanae near the posterior end of the middle turbinates and on the posterior and lateral surfaces of the vomer—all locations inaccessible to operative procedures. Proof that these nodules are often the cause of recurrent symptoms is afforded by histologic demonstration that they have crypts and by the marked clinical improvement that follows their treatment with a radium applicator. The action of irradiation, in the dosage used, is to stop mitosis in the cells which have absorbed the rays. After adequate irradiation no new lymphocytes are formed to replace those lost by the normal dissolution process. The nodule of lymphoid tissue gradually shrinks, the crypts become flattened out or obliterated, and no favorable places remain in the nasopharynx in which the virus can lodge and remain undisturbed by the normal flow of mucus for a long enough time to allow it to enter cells.

We do not mean to suggest that the nasopharynx is the only portal of entry of the common cold virus, but certainly the first symptoms after experimental inoculation of the nasal passages of chimpanzees or human volunteers with the common cold virus is hyperemia and irritation in the nasopharynx, not in the nose or the pharynx. These findings suggest that it is possible for the otolaryngologist to do more to prevent recurring virus infections and their complications than either the pediatrician or the internist, whose therapy is limited to vitamins, nasal drops and antibacterial drugs. These measures usually lead to recovery of the acute condition, but in no way protect against a recurrence of a virus infection. The mucous membrane of the ears and the accessary nasal sinuses has a remarkable ability to recover after a single or an occasional infection. It is the oft-repeated, acute bacterial infections that eventually lead to chronic suppuration and necessitate radical operations. These repeated acute bacterial infections often are initiated by a preceding virus infection in the upper air passages. For this reason it is important that the otolaryngologist should do everything he can to lessen the frequency of colds and to prevent recurrence of pyogenic complications. The only known way to accomplish this, even in part, is to examine the nasal passages and the nasopharynx after the acute infection has subsided and, if hyperplastic or chronically infected nodules of lymphoid tissue are seen in the nasopharynx, in the posterior part of the nose or around the eustachian tubes, remove them surgically, or at least change their contour with surgical operation, irradiation, or both.

It is not possible to remove all lymphoid tissue in the nasopharynx at operation and not safe to give the heavy doses of irradiation that would be necessary to remove permanently all lymphoid tissue in the nasopharynx. Years of experience have shown that small doses are effective in reducing the size and contour of lymphoid nodules without injury to other cells. The safe way to use a nasopharyngeal applicator is to be conservative and satisfied to relieve symptoms as they arise, and not try to obtain a permanent cure with one or more large doses. It is not possible always to cure a patient permanently with one operation, such as removal of tonsils and adenoids, or with one course of antibiotics. The same reasoning holds true for irradiation. Irradiation is a therapeutic measure that is of value in selected cases when used conservatively and safely in conjunction with surgery, antibiotics or other measures.

Our discussion of acute sinus infection will be limited to treatment of the frontal sinuses. The nasofrontal duct is small and sub-

ject to anatomical variations that often make it difficult to catheterize and irrigate the frontal sinuses. Drainage is essential in order to relieve pain and prevent complications, such as osteomyelitis, orbital abscess, brain abscess or meningitis. If, in the presence of an acute infection, the mucous membrane lining the nasofrontal duct is injured in trying to irrigate or start drainage by the nasal route, often the end result is granulations, scar tissue and a chronic sinusitis that necessitates surgical operations. As in acute mastoid infections, we cannot always depend on the administration of antibiotics to cure an acute frontal sinus infection, unless there is also free drainage of pus. The obstruction to drainage in acute frontal sinusitis is not due solely to edema of the mucous membrane at the nasal end of the duct that may be relieved by local application of vasoconstrictor drugs and heat. From our experience in opening the frontal sinus through a small incision in the eyebrow in 88 cases of acute sinusitis, we have learned that the chief cause of obstruction is thick, fibrinous plugs of mucopurulent secretion in the sinus cavity. We must remember that the first changes in the sinus following obstruction of the duct are absorption of air in the cavity, congestion of blood vessels and edema of the lining mucous membrane—an aerosinusitis. If bacteria are present, an abscess results. For prompt relief of pain, prevention of complications and avoidance of chronic sinusitis, free drainage is necessary, and it should not be delayed.

For these reasons we have drained the frontal sinus through an external incision in all patients coming to the hospital with severe pain, tenderness on light percussion over the affected frontal sinus and changes on transillumination and x-ray films. If the pain is very severe, we promptly give penicillin intramuscularly and proceed immediately with the operation. If the pain is less severe, we give both penicillin and sulfamerazine and use heat and vasoconstrictors locally. In 16 of 104 patients this conservative treatment was followed by recovery without external drainage. In the remaining 88 patients the infected frontal sinus was opened through an incision about ½ inch in length, which is made through the unshaven eyebrow just above the inner canthus of the eye. The patient is anesthetized with sodium pentothal and, with the eyes protected with a sterile, thin, flat, circular piece of cotton moistened with sodium chloride solution and covered with vaseline, the eyebrow and surrounding skin is washed with soap and water, then ether and zephiran. This method of preparation avoids the necessity of shaving the eyebrow, which when removed is very conspicuous and embarrassing to the patient for several weeks after he has recovered from the infection.

The incision through the skin and periosteum is just long enough to permit an opening ⅛ inch in diameter to be made in the underlying bone. Great care is taken to avoid traumatizing the soft tissue and periosteum with retractors or clamps. The opening in the bone is made with a perforator and burr. The exposed mucous membrane of the frontal sinus often balloons out into the incision because of the pressure of pus and gases in the sinus. The bulging mucous membrane is incised with a knife and the pus removed with a small suction tube, no larger than a eustachian catheter. The mucous membrane lining the sinus should not be removed or injured. No gauge or curettes are used in the sinus, and no effort is made to probe or enlarge the nasofrontal duct. If uninjured, the mucous membrane will soon become normal, as it does in the nose or pharynx after an acute infection, but if scraped or wiped with gauze, granulations and scar tissue are likely to prevent complete recovery.

After the pus has been removed from the sinus with suction, the cavity is filled with penicillin (1000 units per cc. in sodium chloride solution) or with a tyrothricin suspension (1 cc. of the stock solution in 25 cc. of distilled water). A short piece of soft rubber tubing is put in the sinus to keep the skin edges apart. This is removed 24 hours later and through this opening the sinus is gently irrigated, usually with warm sodium chloride solution. No drain is replaced, since this procedure reinfects the cavity with skin organisms. To prevent closure of the skin incision, the edges are lightly painted with carbolic acid once a day, care being taken to prevent any of the acid from getting into the sinus. The sinus is irrigated with saline once a day, and usually after three or four days it begins to flow through the nasofrontal duct and out through the nose. The skin incision is then approximated and as a rule no further treatment of any kind is necessary. We have employed this technique for several years and, if the cases are carefully selected, the results have been most satisfactory.[4] We have had no complicating osteomyelitis or cellulitis; on the contrary the drainage prevents both acute complications and the development of a chronic sinusitis. If the acute infection that brings the patient to the hospital is superimposed on a long-standing chronic infection, the external incision is used to obtain drainage, relieve pain and identify the infecting organisms, but as a rule more extensive surgical procedures are necessary to cure the patient.

SUMMARY

Acute frontal sinusitis is a painful and potentially dangerous malady. To relieve pain, lessen the danger of osteomyelitis and other

complications, we advocate external incision and drainage in selected cases. This is in addition to the administration of antibiotics. The chief advantage of external drainage is that it makes unnecessary intranasal instrumentation, which not only is very painful but damages the acutely infected, edematous mucous membrane of the nasofrontal duct. The less the infected mucosa in this area is handled, the more likely it is to return to normal when the acute process subsides. Nothing should be done, while trying to tide the patient over the acute period, that will traumatize the infected, edematous mucous membrane of the nasofrontal duct, since this may lead to the formation of granulations and scar tissue that permanently impair drainage, and to the development of a chronic sinusitis. It is far easier to prevent a chronic sinus infection than it is to cure it.

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XXXIII

MONILIASIS OF THE LARYNX: REPORT OF TWO CASES

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The monilia and blastomyces are classified by Henrici[1] in a transitional group between the true molds and the yeasts. Their structural characteristics and cultural reactions seem to vary at different stages of growth so that at one period they may appear to be a mold and at another a yeast. The same organism may be put in varying genera by different mycologists. These variations in classification and identification make the differential diagnosis between moniliasis and blastomycosis rather uncertain and a terminology rich in synonyms adds further to the confusion of nomenclature. However, the specific identifications of these mycotic infections of the larynx are not so important from a clinical standpoint, as the pattern of symptoms, physical findings and treatment is much the same for both of them. The importance of the diagnosis of moniliasis of the larynx resides in its differentiation from diseases with a far higher incidence, such as cancer, tuberculosis, syphilis and acute simple laryngitis, rather than in distinguishing it from blastomycosis.

Thrush, or the monilial infection of the oral mucosa, is of relatively common occurrence, bronchomoniliasis, which so often simulates chronic pulmonary tuberculosis, is being recognized with increasing frequency, but moniliasis of the larynx is rarely referred to in the literature. Two cases of monilial infection of the larynx are here reported. In one, an infiltrative granuloma mimics a cancer, and the other may be classified as an acute or subacute laryngitis.

CASE 1.—Mrs. T. D. H., age 48, was referred with the complaint of hoarseness. The onset of hoarseness occurred about eight months previously and during the past three months had become progressively worse. Pain in the left ear was first noted four months ago and recently has been increasingly persistent and uncomfortable.

Read before the meeting of the American Laryngological Association, Hot Springs, Va., April 15, 1948.

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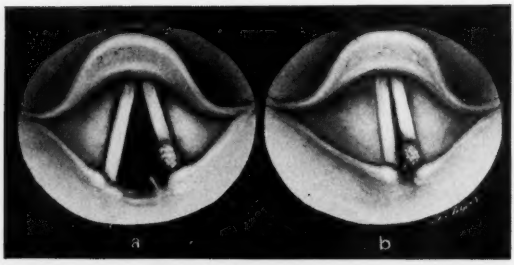

Fig. 1.—Schema of the larynx; *a*, inspiration, *b*, phonation.

Pain on swallowing has been worse during the past month. The previous history referable to the presenting complaint was that a diagnosis of chronic pulmonary tuberculosis had been made four years ago, but the referring physician did not agree with this. A persistent cough developed one year ago from a chronic bronchitis.

The examination of the larynx demonstrated one tumor on the left vocal cord, another on the interarytenoidal mucosa, and no membrane. The left vocal cord was fixed in abduction and a warty, slightly pink, tumor occupied its posterior third with a small extension to the interarytenoidal mucosa. The tumor on the interarytenoidal mucosa was apparently a pedunculated papilloma. The persistent hoarseness, referred pain to the ear, dysphagia, cough and a tumor on a fixed vocal cord as shown in Fig. 1, suggested a differential diagnosis between cancer, tuberculosis and syphilis. The radiologist reported that the x-ray film of the chest showed bronchovascular mottling of both lungs, no tuberculosis, no cancer, but findings indicating a monilial infection (Fig. 2). The clinical pathologist reported that the serological tests for syphilis were negative and that the sputum examination was negative for Bacillus tuberculosis, but showed a profuse amount of monilia albicans (Fig. 3).

On the basis of these reports a preliminary diagnosis of cancer of the larynx was made and a specimen taken for pathological examination. The report on the specimen (Fig. 4) was chronic inflammatory tissue, nonspecific, with no evidence of malignancy. A second biopsy was done five days after the first to confirm the pathological report. The report on the second specimen was the same as on the first.

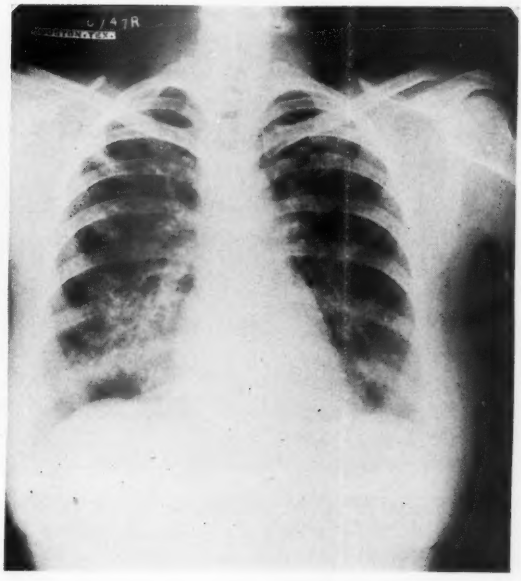

Fig. 2.—Roentgenogram showing bronchomoniliasis.

The postoperative reaction of the larynx after the second biopsy was quite severe and the infection could not be controlled by topical treatment. Since streptomycin is effective in tuberculous granuloma of the larynx, it was selected for the treatment of the monilial infiltrative granuloma. Intramuscular injections of streptomycin, 1 gm. per day, were administered for three days. On the fifth day after starting streptomycin, the infection was greatly improved, the left vocal cord was freely mobile, only a small remnant of the granuloma or "tumor" remained on the left vocal cord and the granuloma on the interarytenoidal mucosa had disappeared. A second series of streptomycin injections was begun on the sixth day and continued for three days. On the tenth day, the vocal cords were freely mobile and healed with no evidence of a granuloma, but they were still hyperemic and thick. Monilia being present in the sputum, potassium iodide was prescribed in increasing dosage. The voice was clear six weeks later.

CASE 2.—Mr. J. L. C., age 54, had a complaint of malaise and hoarseness. The onset occurred two weeks previously without fever.

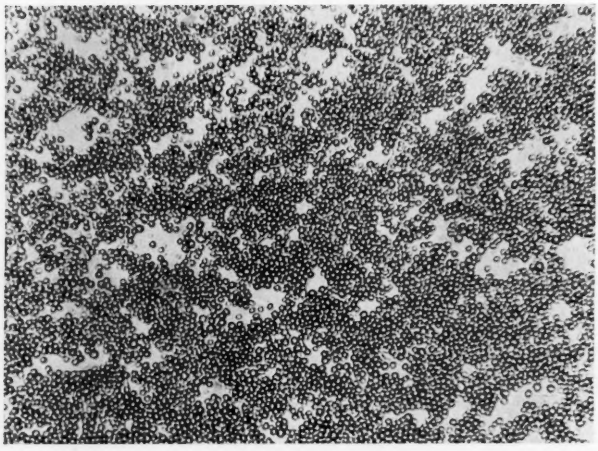

Fig. 3.—Photomicrograph of monilia albicans; two weeks' growth. X 237.

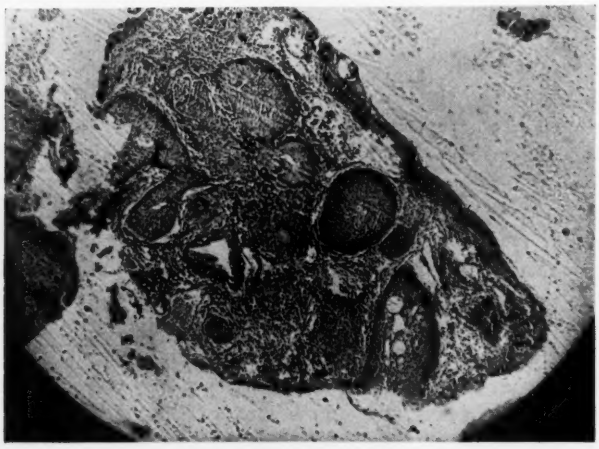

Fig. 4.—Photomicrograph of monilial granuloma of the larynx. X 54.

The voice was husky, tired rapidly and became hoarse on talking for a short time. He has had a cough which he attributed to cigarettes and recently an increasing amount of sputum. The examination of the larynx demonstrated an acute laryngitis without any membrane. The vocal cords were mobile, met in the midline on phonation and the mucosa was hyperemic and thickened in small patches.

The acute laryngitis was treated systemically and topically. The laryngitis under this therapy rapidly became worse. The patches of hyperemic and thickened mucosa extended and coalesced with each other, so that the vocal cords took on a red velvety appearance. The x-ray film was reported to be negative for chronic pulmonary tuberculosis. The serological tests for syphilis were negative. The sputum examination was negative for bacillus tuberculosis, but showed a large amount of monilia albicans. After the previous experience with monilia, it was not regarded as merely a concomitant finding. Intramuscular injections of streptomycin, 1.5 gm. per day, were administered as in Case 1. The acute laryngitis was much improved on the tenth day; but a few tiny patches of hyperemic and thickened mucosa remained on the vocal cords and monilia persisted in the sputum. Potassium iodide in increasing amounts was exhibited but was discontinued on account of the patient's poor tolerance. Liquor iodi compositus (Lugol's solution) in an increasing dosage was effective in clearing the small red patches of thickened mucosa from the vocal cords and the monilia from the sputum. The hoarseness and cough were relieved.

No conclusions, of course, can be based on two cases, but the observations here reported may be summarized as follows:

1. Moniliasis of the larynx may occur as an acute or chronic infection.

2. A monilial infiltrative granuloma of the larynx is rare, and it may mimic cancer.

3. Streptomycin controls an acute monilial infection of the larynx and causes the rapid disappearance of a granuloma, but in the dosage here reported does not clear monilia from the sputum.

4. Liquor iodi compositus ((Lugol's solution) is effective in treating moniliasis and clearing monilia from the sputum.

5. The incidence of acute laryngitis due to monilia is unknown, but like bronchomoniliasis it may be recognized with increasing frequency.

6. Monilia in the sputum does not necessarily demonstrate it to be the invading organism.

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XXXIV

CANCER OF THE LARYNX

A STATISTICAL STUDY

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This is a report of 500 consecutive cases of cancer of the larynx seen at the Massachusetts Eye and Ear Infirmary from 1930 to 1945, inclusive, and treated either at the Infirmary or in the Radiological Department of the Massachusetts General Hospital. Included in the report are 46 patients who were seen, treated and followed as private patients.

Of the total of 500 patients, there were 28 who were seen for diagnosis only and who were treated elsewhere; 23 who refused treatment; and 32 who were treated but could not be traced. Therefore 418 cases of cancer of the larynx were followed from the hospital records and by correspondence with the patient, with their families, their family physician or with their town or city clerks.

Cancer of the larynx is a disease of males. In this series 92 per cent were male patients and only 8 per cent female. The average age of the male patients was 60 years while that of the female patients was 40 years. The youngest patient was a young man of 20 years.

Tracheotomy was required in 80 cases, 25 of these were intrinsic cancer and 55 were extrinsic. In 46 cases tracheotomy was urgent and was done preliminary to treatment, while in 34 cases tracheotomy was necessary either during or soon after external irradiation.

This study was financed by a grant from The American Cancer Society, Massachusetts Division.

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Presented before The American Laryngological Association, Hot Springs, Va., April 15, 1948.

TYPE OF LESION

Carcinoma Grade 1	35	or	7%
Carcinoma Grade 2	210	or	42%
Carcinoma Grade 3	140	or	28%
Carcinoma Grade 4	25	or	5%
Ungraded	55	or	11%
Clinical Carcinoma	25	or	5%
Miscellaneous	10	or	2%

The terms intrinsic and extrinsic cancer are not standardized and convey a different meaning to each observer. Until these terms are standardized it would be better to describe the lesion as involving an anatomical structure, such as the true vocal cord, the laryngeal ventricle, or the false cord, rather than by the use of an indefinite term. Our own interpretation of the term "intrinsic" is that of an involvement of the lumen of the larynx from the laryngeal surface of the aryepiglottic fold down, while that of "extrinsic cancer" is that of involvement of extralaryngeal structures, such as the pyriform sinus, the mouth of the esophagus, or the thyroid cartilages.

With our present day knowledge of cancer we have but two methods of treatment that have stood the test of time, namely, surgery to remove the cancer, and irradiation to destroy it—or a combination of the two. It is the selection of the type of treatment for the individual case (and each case must be individualized) that calls for experience and wise judgment. In this series the records show the type of treatment selected to have been:

External Irradiation	246 cases
Radium with Other Treatment	14 cases
Surgery or Surgery with Irradiation	158 cases

The treatment of cancer of the larynx by external irradiation has not been uniform during the past 15 years. Prior to 1936, the amount of irradiation was usually less than 5000 r and was divided over a period of two or three months. During the past five or six years the technique of therapy, as practiced by the radiologists of the Massachusetts General Hospital, is to give a total dose of at least 6000 r and more often of 7000 r if tolerated by the patient, and additional radiation for extension of the disease and cervical metastasis. The period is four weeks with a daily dosage of 300 r to one

lateral field the following day. In contrast is the technique of Cutler[1] who gives from 6000 r to 6500 r to one portal in 18 days, giving two treatments daily.

With our present method of treatment as carried out at this hospital one would expect to see a higher percentage of cures in the latter years compared to the patients treated during the period of 1930 to 1940. By dividing our series into three groups of five years each and analyzing each group the results of external irradiation are disappointing.

CANCER TREATED WITH EXTERNAL IRRADIATION

YEARS	CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
1930-1935	24	18 - 75%	6 - 25%	
1935-1940	75	58 - 77%	17 - 22.6%	
1941-1945	147	104 - 70%	5 - 3%	38 - 27%

If we take the patients seen and treated during the years of 1930 to 1940 and therefore observed for five years or more, we find that of 99 patients with laryngeal cancer 76 or 76 per cent are dead and that 23 or 23% have a five-year cure of their disease. Since many of the patients seen in the period of 1941 to 1945 have not been under observation or treatment for five years, it is shocking to note that of 147 patients 104 or 70 per cent are already dead.

It is also interesting to note that the number of patients treated by external irradiation during 1935-1940 is three times the number so treated during 1930-1935; that during 1941-1945 twice the number were so treated than during 1935-1940. About 1935 when the first supra-voltage x-ray was installed at the Huntington Memorial Hospital for Cancer Research, it was predicted that supra-voltage irradiation would replace surgery for cancer of the larynx. We shared in that enthusiasm and consequently referred many patients with cancer of the larynx, ideal cases for surgery, for treatment by irradiation. When the Huntington Hospital closed in 1940, the patients and their records were transferred to the Tumor Clinic of the Massachusetts General Hospital and have been accessible for this study. With the further development of supra-radiation to the 3-million volt machine, our interests in the selection of patients for irradiation has continued, as we have been anxious to determine its value on other than the hopeless case. From our experience we are now of the opinion that:

1. In the hopeless case of cancer of the larynx, external irradiation is not palliative and often hastens the demise of the patient.

2. In circumscribed local lesions of the vocal cord, without fixation, five-year cures have been obtained in 46 per cent of a small series of cases.

3. Regression of the lesion does not signify a cure; the patient must be kept under close observation for at least five years.

4. The so-called trial period of irradiation is a fallacy. A full curative dose of external irradiation is necessary to determine radiosensitivity.

RESULTS FROM IRRADIATION ACCORDING TO SITE OF LESION AND EXTENSION

SITE	CASES	DEAD	5-YR.-CURE	LESS THAN 5 YRS.
Laryngeal	72	66%	17%	19%
Epiglottic	38	71%	19%	20%
Subglottic	15	67%	13%	20%
Hypopharynx	66	79%	4%	17%
Tongue	11	91%	0	9%
Metastasis to Neck	111	85%	4%	11%

When the disease is limited to the intrinsic larynx, particularly the vocal cords or the supraglottic structures, the chances of a cure by external irradiation are best. As the lesion extends beyond the larynx, the prognosis grows definitely worse. It is almost nil when the extension reaches the tongue and 85 per cent of our patients with glandular involvement are dead. Actually the percentage will be higher than 85 per cent when the final result of those now under observation less than five years is ascertained.

The time to cure cancer is the first time that you attempt to do anything about it whether by surgery or by irradiation. Surgery should be radical through normal tissue to remove the disease. The problem is not how little you can remove, but how much.

With irradiation the dosage should be to the extent of skin tolerance; when the first series of adequate irradiations fails to produce a regression of the lesion, subsequent treatment is of little if any avail.

RELATION BETWEEN GRADE AND RESULT FROM IRRADIATION

GRADE	CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
Grade 1 & 2	80	61%	19%	20%
Grade 3 & 4	104	72%	8%	20%

The general opinion among many surgeons regarding choice of treatment for carcinoma of the larynx depends upon the grade and histological picture of the tumor. Carcinoma grades 1 and 2 are generally considered radioresistant and grades 3 and 4 radiosensitive. Jackson and Jackson[2] state: "Tumors of grade 1 and 2 that used to be considered radio-resistant are apt to be more radio-curable than the less differentiated tumors of higher grades. These latter tumors of grade 3 and 4 carry a poor prognosis with any method of treatment." According to Cutler[1] radiosensitivity depends upon the gross and microscopic picture and the site of the lesion. The choice of proper treatment should not depend upon grading alone. Cancer of one cord is radiosensitive only if treated early before infiltration and fixation. Cancer arising from the false cords and the epiglottis is radiosensitive. Any cancer that infiltrates and fixes these structures is radioresistant.

In this series, the results obtained did not depend upon the grading of the tumor alone, but depended to a greater extent on the extension of the disease. The results of external irradiation were better than two to one in carcinoma of grades 1 and 2 than in grades 3 and 4.

CORDAL CANCER (IDEAL FOR SURGERY) TREATED BY IRRADIATION

CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
11	3 - 27%	5 - 46%	3 - 27%

These 11 cases were considered ideal for surgery in that the lesion was localized without fixation of the cord. Irradiation gave a five-year cure in 46 per cent of the cases. With the final result of the three cases that are still under observation this percentage without a doubt will be better.

The usual procedure when a patient presents himself with malignant disease at the Massachusetts Eye and Ear Infirmary is to admit the patient to the hospital and after a thorough study, to get a biopsy specimen and obtain the microscopic report. The patient is then referred to the Tumor Clinic where he is examined by a group, including an otolaryngologist, a surgeon, and a radiologist. There the case is discussed and the treatment decided. Previous to 1940, when surgery was recommended, the patient was handled by the Surgeon on Service. As a consequence too many operators without Tumor Clinic experience were doing cancer surgery. Since 1940 all tumor surgery is done by one of us (L. A. S.) or done under our direction.

The laryngeal cancer ideal for thyrotomy or laryngofissure is a localized lesion of the anterior third or the middle of a vocal cord, without involvement of the anterior commissure or extension to the vocal process, preferably of low grade, and without fixation of the cord.

LARYNGEAL CANCER TREATED BY LARYNGOFISSURE

YEARS	CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
1930-1940	31	13 - 42%	18 - 58%	
1941-1945	28	8 - 18%	2 - 8%	18 - 74%

From an analysis of our records only 39 cases were ideal for laryngofissure. Eighteen patients should not have had a laryngofissure because of cord fixation, extension to the arytenoid or by reason of subglottic extension. Of these 18 only one was cured by surgery, two had laryngectomies after laryngofissure failure; four have been under observation less than five years; three have been cured by irradiation and eight are dead. Out of the 20 patients who died after laryngofissure there were two who were operative mortalities: one from pulmonary hemorrhage from an old tuberculosis lesion on the eighth postoperative day and the second from a postoperative lung abscess (1934). Eight should have had a laryngectomy instead of a laryngofissure. Ten were apparently well chosen, but recurrences developed.

A review of our failures with laryngofissure reveals that the recurrences have mostly occurred in those cases in which the cancer had reached the anterior commissure, had crossed to the other cord or had reached the vocal process. For the anterior commissure lesion, we have followed the Jackson anterior commissure technique, but as with all laryngofissures, we have used the diathermic scalpel and snare for incision and removal of the soft parts of the lumen of the larynx. The cut surface is also dessicated by electrocoagulation.

We still use the nasal feeding tube for 24 to 48 hours, and from experience advise its insertion before, rather than after, the operation when the swallowing reflex is abolished by the anesthetization of the larynx.

LARYNGECTOMY

YEARS	CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
1930-1940	44	16 - 36%	28 - 64%	
1940-1945	53	14 - 26%	2 - 4%	37 - 70%

This group of laryngectomized patients can be further broken down into those who were treated only by surgery and those in whom the extension of the disease made the operative removal questionable and who therefore received external irradiation after surgery

LARYNGECTOMY AND IRRADIATION

	CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
Laryngectomy	63	14 - 22%	19 - 30%	30 - 48%
Laryngectomy and X-ray	34	16 - 47%	11 - 33%	7 - 20%

Our over-all statistics on laryngectomy of the five-year or longer group show a survival rate of 64 per cent, while that of New[3] is 60.2 per cent.

The psychology of a surgeon dealing with cancer should be radical. The problem is how much can be removed and not how little. When a patient is faced with the loss of his larynx, he is not interested any longer in conservative surgery. The routes of extension of cancer of the larynx have been well demonstrated by Taylor and Nathanson;[4] and the importance of removing the pre-epiglottic space has been emphasized by Bisi.[5]

In total laryngectomies, we have always used the "T" incision or the "U" incision of Gluck-Soerensen. The sternohyoid muscles, and at times the anterior belly of the omohyoids are removed with the hyoid bone or the body of the hyoid bone.

The pretracheal gland on the cricothyroid membrane is always examined microscopically for cancer. The pretracheal muscles are removed since they are rich in lymphatics and are in close relation to the pretracheal gland. We have found metastatic cancer in the sternohyoid muscle when, on gross inspection, it could not be detected. The thyrohyoid muscle is removed with the larynx.

The carotid sheaths are always exposed and examined for involved glands, and should they be found a radical neck dissection is done along with the laryngectomy. In cases of laryngeal cancer with unilateral cervical metastasis both Sylvestre[6] and Brunschwig[7] have demonstrated not only their operability but an encouraging curability by radical neck dissection and laryngectomy.

We have experimented with closure without drainage and have obtained a surprising number of primary closures. The fistulas, since the advent of the sulfonamides and the antibiotics, are not a problem.

In this series of 97 laryngectomies there were four operative deaths: one from a lung abscess, one from a peri-esophageal abscess; two were cardiac deaths, one patient dying on the eighteenth day, the other suddenly on the third day. Four patients died suddenly, either at home or on the street. One patient (female) died from pneumonia three years after the operation; a diabetic survived ten years. Ten patients died of extrinsic disease and although 12 were by all standards good risks, the operations were frank failures.

When external irradiation fails to control the disease, surgery can still be done although the healing may be delayed. In this series laryngofissure was done in four cases and laryngectomy in eight after external irradiation failed to control the disease. Two of the laryngectomies were done on patients for recurrences, occurring after three years of apparent cure by irradiation.

LARYNGECTOMY AFTER X-RAY FAILURE

CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
8	2	2	4

In 19 cases of distant metastases, the following organs were involved: lung 11, brain 3, spine 2, and one each of knee, hip, jaw, prostate and skin of the chest. In one patient whose larynx was free of disease for seven years, the lung and thigh showed metastases. In another, after laryngofissure in 1935, laryngeal recurrence appeared in 1941, and after treatment by x-ray there was regression of the growth. Metastasis in the lung developed in 1943 and was treated by lobectomy; the patient died of postoperative empyema.

RESULTS OF SURGERY ON GRADE 3 AND 4 CARCINOMA

	CASES	DEAD	5-YR. CURE	LESS THAN 5 YRS.
Laryngofissure	4	3	0	1
Laryngectomy	7	3	1	3
Laryngectomy and X-ray	11	4	4	3

It is difficult to draw conclusions from this small series. However, it is evident that surgery in well chosen cases of cancer of high grade malignancy has some chance of success. Since high grade malignant disease is difficult to cure by any method of treatment, we fell that combined treatment—surgery and external irradiation—offers the patient the best chance of life.

The surgeon's duty to his patient does not end with the discharge of the patient. At least a five-year follow-up is necessary to detect early recurrence. The laryngectomized patient should be rehabilitated by the development of a voice. We have followed the work of McCall[8] in preoperative training, and by the establishment of a daily speech class.

Many of our patients have developed a good esophageal voice within two weeks following their laryngectomy, while others require months. Many of our patients have come from other cities and many have been operated on from one to four years ago without any attempt being made in rehabilitation by either an esophageal voice or the artificial larynx. In our clinic in the past year over 100 patients have been taught to speak with the so-called esophageal voice.

SUMMARY

A total of 418 cases of carcinoma of the larynx were studied, the treatment classified and evaluated. Of these 418 patients, 246 were treated by external irradiation. Twelve patients underwent surgery after external irradiation failed to control the disease.

The site of the lesion and the extension of the disease are of more importance in the proper choice of treatment than the grading of the tumor. Carcinoma of grades 1 and 2 is more radio-curable and surgery-curable than are the more anaplastic types of growth.

Limited lesions of grades 3 and 4 are best treated by surgery followed by postoperative external irradiation. Laryngofissure is the operation of choice for early cancer limited to one cord, without fixation. Extension of the growth to the anterior tip of the opposite cord is still amenable to laryngofissure by wider dissection. Any fixation or posterior extension to the arytenoid, even without fixation, contraindicates laryngofissure.

Early unfixed cordal lesions are 46 per cent radio-curable by concentrated external irradiation. With the extension of the disease to the subglottic space and with cord fixation laryngectomy is the treatment of choice and not external irradiation. Patients with borderline cases, cases which some observers call extrinsic, can be successfully treated by surgery and irradiation.

In a group of poorly selected cases five-year cures were obtained by laryngofissure in 58 per cent. Total laryngectomy on a series of patients operated on between 1930 and 1940, and therefore followed over five years resulted in a 64 per cent survival.

With the patient under close observation, a full course of irradiation is justifiable with the distinct understanding that unless the tumor shows evidence of regression, surgery will be instituted.

Early diagnosis and repeated biopsies of suspicious lesions until the diagnosis is absolutely established can never be overemphasized, as any delay will make the disease less and less favorable for cure by any of our methods of treatment.

243 CHARLES STREET.

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XXXV

SKULL ROENTGENOGRAMS OF INTEREST TO THE OTOLARYNGOLOGIST

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The otolaryngologist in viewing sinus roentgenograms, whether obtained in his office or elsewhere, will usually confine his observations to the paranasal sinuses. However, other less noticeable conditions of the calvarium and its contents are occasionally visualized and will attract the eye of the interested and curious. A review of

Fig. 1.—Calcification of the Falx Cerebri.

some pathologic conditions and congenital anomalies of the skull and intracranial contents which can be recognized by the otolaryngologist in x-ray films of the sinuses seems worthwhile.

Presented before the Southern Section Meeting of the American Laryngological, Rhinological and Otological Society, New Orleans, La., Jan. 23, 1948.

Fig. 2.—Luetic Osteomyelitis.

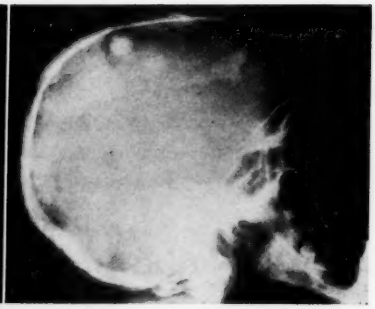

Fig. 3.—Monophasic Stage of Paget's Disease.

Fig. 4.—Diphasic Stage of Paget's Disease.

Fig. 5.—Osteoma of the Parietal Region.

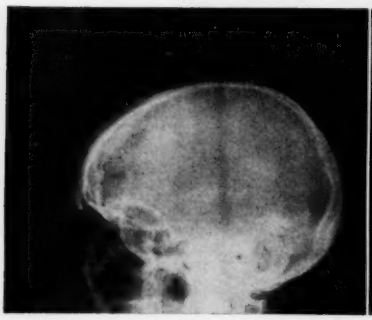

Fig. 6.—Hyperostosis of the Inner Table of the Frontal Bone.

Fig. 7.—Erythroblastic Anemia.

Most otolaryngologists realize the desirability of routine roentgenograms of the sinuses in office practice. The value of the roentgenograms is enhanced by including the entire skull in the examination. A knowledge of some of the pathological lesions of the skull is especially helpful when an expert radiologist is not immediately available.

Two cases illustrate the object of this presentation. The first patient was a middle-aged woman complaining of headache. Repeated clinical examinations by several specialists had been negative and she had been classified as a neurotic. Routine roentgenograms of the sinuses were negative; on the lateral view a marked hyperostosis frontalis interna was observed. The headaches were then attributed in part to an organic lesion; the patient was given an explanation as to a possible cause of the pain and endocrine therapy was started. She obtained considerable relief from this approach to her problem.

The second illustrative case was told to me by a colleague. His patient also complained of headaches. The sinus roentgenograms were negative; the lateral view of the skull disclosed a fracture line in the parietal region. On careful questioning a forgotten head injury was recalled, and on surgical exploration a subdural hematoma was found. If the x-ray examination had been restricted to the sinuses and the anterior portion of the skull, this lesion would have been overlooked.

Needless to say the roentgenograms should be of good quality, with good definition and correct positioning. The lateral view of the sinuses should not be confined to the paranasal sinuses but should include the entire skull. From the lateral view most of the extrasinus lesions are identified.

In the x-ray examination of the skull a knowledge of the normal variations in the skull architecture is important. The appearance of the diploic structure, convolutional and vascular markings, normal size and shape of the sella turcica, and depressions on the inner table of the skull should be known. Fig. 1, for instance, illustrates a calcification of the falx cerebri, a normal finding. In another instance a skull roentgenogram showed a coarse granulated diploic structure which suggested hyperparathyroid disease. Negative laboratory tests placed this case in the class of a normal variation in skull structure.

When a routine is established, one can rapidly survey the rest of the skull after the paranasal sinuses have received initial attention.

Fig. 8.—Mediterranean Anemia.

Fig. 9.—Meningioma of the Sphenoidal Ridge.

Fig. 10.—Metastasis from Carcinoma of the Breast.

Fig. 11.—Metastasis from a Chordoma.

Fig. 12.—Multiple Myeloma.

Fig. 13.—Hyperparathyroidism.

The plan of inspection of the skull roentgenograms should include:

1. A consideration of the vault.
2. A consideration of the region of the sella turcica.
3. A consideration of the intracranial cavity for calcified shadows.

Consideration of the Vault. Aside from acute osteomyelitis of the frontal bone, with which the otolaryngologist is familiar, the cranial vault can be invaded by many specific, metastatic and metabolic diseases. An unusually extensive luetic osteomyelitis of the skull is shown in Fig. 2.

The osseous skull changes in Paget's disease provide a startling appearance in the roentgenogram. Fig. 3 illustrates the monophasic stage of the disease, the phase of osteoporosis circumscripta, showing the areas of rarefaction and no evidence of bone regeneration. Fig. 4 depicts the diphasic stage of Paget's disease, the stage of bone regeneration. Paget's disease is usually seen in the older age group and is often associated with deafness. The condition is characterized by an enlargement of the skull which is due to an accelerated resorption of the areas of bone followed by excessive replacement with new bone of a porous nature. The process is most marked in the outer table and diploe.

A common tumor involving the calvarium, nasal bones and nasal accessory sinuses is the osteoma. The osteoma is a benign, localized, bony new growth, and may be one of two types, the spongy or the ivory-like sclerotic type. The appearance on the roentgenogram depends on the type of growth; the outer table is usually involved and is obscured by the tumor. In contradistinction to the hyperostosis associated with the underlying meningioma the outer table is visible. Fig. 5 depicts an osteoma involving the parietal bone.

Also to be differentiated from the osteoma is the interesting entity not infrequently seen, hyperostosis of the inner table of the skull, which is illustrated in Fig. 6. The newly formed bone on the inner table of the frontal bone is seen bilaterally and is symmetrical. Hyperostosis frontalis interna is often associated with neuropsychiatric and endocrine symptoms, the exact status of which is unknown. When associated with the endocrine symptoms of obesity, virilism and menstrual disorders it is called the syndrome of Morgagni; when associated with the neuropsychiatric symptoms of headache, muscular weakness, paretic muscles, personality changes and dementia it is called the syndrome of Stewart Morel.

Fig. 14.—Xanthomatosis.

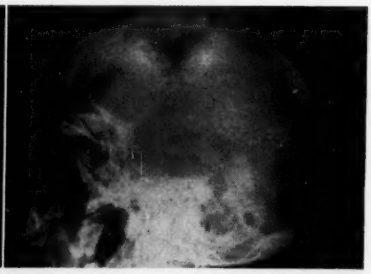

Fig. 15.—Cholesteatoma.

Fig. 16.—Hypophysial Tumor.

Fig. 17.—Suprasellar Cyst.

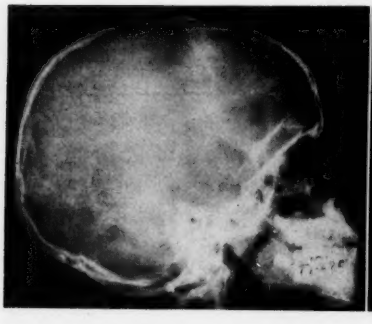

Fig. 18.—Erosion of the Dorsum Sellae.

Fig. 19.—Increased Intracranial Pressure.

The otolaryngologist may be confronted with this condition in a patient, usually a woman in the menopausal age, complaining of headache and vertigo. Estrogenic therapy seems to bring a certain measure of relief.

The association of localized areas of hyperostosis and an underlying meningioma in 50 per cent of cases makes a routine inspection of the areas of predilection important. The sites of predilection of these tumors are the sagittal region of the cortex, the basal region, the petrous ridge, the olfactory groove, the lesser and great wings of the sphenoid and the roof of the orbit. The roentgenogram is characterized by a perpendicular striation and a thickening of the vault involving the inner and outer table and the diploe. This appearance may possibly be confused with certain types of congenital anemias, erythroblastic anemias or Cooley's anemia, as illustrated in Figs. 7 and 8. The appearance here is a widening of the diploic spaces, atrophy of the outer table and the "sunburst" type of striation.

In the basal regions adjacent to the tuberculum sellae, the hyperostosis associated with the meningioma is in the nature of diffuse bony thickening or proliferation. This is illustrated in Fig. 9, a sphenoidal ridge meningioma. Recall that the osteoma is a localized bony thickening; the meningioma more diffuse.

The finding of solitary or multiple defects or rarefactions in the vault is startling and often perplexing. Figs. 10-15 illustrate some common and some unusual conditions of this group.

In Fig. 10 we see multiple irregular areas of bone destruction characteristic of metastasis in the skull, in this case from carcinoma of the breast. The primary sources of metastases are the breast, thyroid, lung, kidney, prostate and rectum. The diploic space is first involved, later the inner and outer tables are eroded. Fig. 11 is a metastatic skull lesion from a chordoma.

Multiple myeloma is illustrated in Fig. 12. The x-ray description of this lesion is a punched-out defect in the skull, a "moth-eaten skull." Multiple myeloma is a neoplastic-like growth involving the ribs, spine, long bones and skull, and is diagnosed by chemical changes in the blood and urine and by sternal puncture.

In hyperparathyroidism the x-ray appearance of the skull is a mottling and demineralization of the bone. Blood calcium and phosphorus studies are diagnostic. Fig. 13 illustrates this condition.

The larger skull defects are seen in xanthomatosis, primary cholesteatoma, or epidermoid tumors and eosinophilic granuloma.

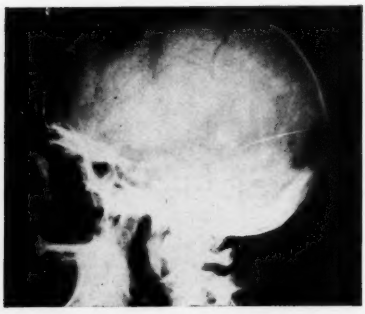

Fig. 20.—Calcified Tumor.

Fig. 21.—Oxycephaly.

Fig. 22.—Craniolacunia.

Xanthomatosis or Christian-Schüller's disease is a syndrome combining defects in the membranous bones, exophthalmos, and diabetes insipidus. The large multiple defects in the skull have distinct, clear-cut, irregular edges. There may be destruction of the sinuses, mastoids, and facial bones (Fig. 14).

In primary cholesteatoma (Fig. 15) the roentgenogram shows variable irregular areas of bone destruction in the parietal region.

Consideration of the Sella Turcica. A survey of the region of the sella turcica is of great interest. An enlargement in the size and depth of the pituitary fossa with erosion of the clinoid processes is diagnostic of a hypophysial tumor as illustrated in Fig. 16. (An incidental finding in this slide is a calcification of the pineal body and the choroid plexus.) A calcified area above the sella is diagnostic of a suprasellar cyst as illustrated in Fig. 17.

Figure 18 shows an erosion of the dorsum sellae which is diagnostic of an expanding lesion near the sella or a dilated third ventricle. The roentgen signs of increased cranial pressure include atrophy of the dorsum sellae, increase in size of the vault, convolution atrophy indicated by increased number and depth of the digital markings, and in children widened or separated sutures. This is illustrated in Fig. 19. (Note the elongated sella.)

All in all the region of the sella is rich in possibilities—tumors (30 per cent of the brain tumors), aneurysms, cysts, and arachnoiditis.

Consideration of Intracranial Calcifications. A routine inspection of the anteroposterior view for pineal calcification is worth while. The pineal body, a midline structure, is calcified in a majority of adult skulls and may be displaced laterally to the opposite side by tumor pressure or to the same side by brain softening. Its displacement also may be a helpful adjunct in the diagnosis of subdural hematoma.

Other physiologic calcifications may be seen in the falx cerebri, choroid plexus, interclinoid and petroclinoid ligaments, and pacchionian granulations. Calcification of the cerebral basal ganglia is seen in encephalitis, tuberous sclerosis, toxoplasmosis and in parathyroid insufficiency. This type of calcification, as seen in the anteroposterior view, is bilateral and symmetrical. In the lateral view, the calcification of the choroid plexus is, of course, more posterior than the calcification of the basal ganglia.

Of most important diagnostic and localizing value is the calcification exhibited by some cerebral tumors. It is said ten per cent of the cerebral tumors calcify, especially the slow growing astrocytomas and oligodendrogliomas. A calcified cerebral tumor is illustrated in Fig. 20.

Figures 21 and 22 show some of the congenital anomalies of the skull. Fig. 21 illustrates the type of craniostenosis known as oxycephaly. Craniostenosis is a malformation of the skull caused by a premature synostosis of one or more of the sutures, and was accompanied in this case by increased intracranial pressure, exophthalmos and optic atrophy. In the more advanced cases the deformity is marked and often accompanied by congenital anomalies elsewhere in the body.

Hypertelorism is characterized by an increased distance between the orbits.

Craniolacunia, Fig. 22, is an anomaly of the developing vault bones, those bones formed from membrane. Abnormal ossification occurs in the form of dense ridges enclosing elliptical areas in which the bone is diminished or absent. It is usually associated with spinal bifida and meningocele.

SUMMARY

The object of this paper is to review some pathologic conditions and congenital anomalies of the skull and intracranial contents which can be recognized as abnormal by the otolaryngologist in routine roentgenograms of the sinuses; he can then refer the patient to a competent roentgenologist for diagnosis. The otolaryngologist is chiefly interested in the x-ray appearance of the nasal accessory sinuses; however, a routine survey of the cranial vault, the pineal body, the sella turcica, and a search for abnormal calcified shadows will occasionally reveal lesions which should not be overlooked.

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XXXVI

INDICATIONS FOR AND END RESULTS OF FENESTRATION SURGERY

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The medical treatment of clinical otosclerosis has to date not produced any significant results. Inasmuch as, in most instances, the deafness is of a progressive type, the patient will eventually be in need of some type of aid. At the present time, if the individual is sufficiently handicapped and has been found to be suitable for fenestration, the choice he must make lies between the selection of a hearing aid or a fenestration. After the patient has been advised as to what can and cannot be accomplished by surgery. the final decision as to whether or not a fenestration is to be done must be made by the patient. The majority of those who have previously worn an aid are anxious to have a fenestration done if they are at all suitable.

The diagnosis of clinical otosclerosis is based on the following points:

1. There is usually a history of familial deafness, but this in itself is not absolutely essential.

2. The deafness is usually progressive. There frequently are, however, periods during which progression apparently ceases. Pregnancy increases the rapidity of progression somewhat in about one-fourth to one-half the instances.

3. While the deafness is usually bilateral, occasional patients are seen in whom the involved ear may have lost considerable hearing while the other ear has essentially normal or near normal hearing. Tinnitus is a frequent occurrence.

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Presented before Minnesota Academy of Ophthalmology nad Otolaryngology, December 12, 1947.

4. The tympanic membrane is usually normal. If in a conduction deafness the tympanic membrane shows considerable scarring, it will be impossible to state whether the deafness is due to otosclerotic stapes fixation or ossicular chain immobility due to an old adhesive otitis. For practical purposes an accurate differentiation is immaterial inasmuch as an adhesive otitis can be helped by fenestration provided the drum is intact and cochlear function is satisfactory.

5. If there is no associated nerve degeneration, the bone conduction acuity as revealed by audiometric testing should be normal or near normal. If the patient has a 40 to 50 db. loss by air conduction, the Rinne test for the 256, 512 and 1024 c.p.s. forks should be negative. The Schwabach test for these forks should be slightly prolonged or normal. For the 2048 frequency the result of the Schwabach test is a little more difficult to determine, but it should be at a fairly good level. If there is any significant degree of nerve degeneration, the audiometric bone conduction threshold and the fork tests will be modified somewhat, particularly for the higher tones. This will be discussed further under the classification for suitability. Bone conduction tests should be done with suitable masking.

It is not always easy to arrive at a decision as to whether the patient with clinical otosclerosis is suitable for surgery. Regardless of how specific one's criteria for suitability may be, there are frequently borderline cases on which an absolute decision one way or the other cannot be made. It is our custom to evaluate the approximate prognosis as to the probable hearing improvement to be expected from surgery and then to let the patient decide whether or not he or she wishes to have a fenestration.

Selection of Cases.—After a diagnosis of clinical otosclerosis has been made and our routine hearing tests completed, we classify the case as to suitability. Each case is classified into one of three groups, namely:

Class A. These are cases in which there is considered to be satisfactory cochlear function as determined by bone conduction audiometry with no level for the 512, 1024 or 2048 frequencies being below 30 db.* The Rinne tests with the 256, 512 and 1024 forks are negative. The Schwabach tests for these forks show a normal or slightly prolonged time as compared to a normal ear. The Schwabach test is normal or only slightly shortened for the 2048 frequency.

*We use the Maico audiometer which gives slightly lower levels for bone conduction than some other audiometers.

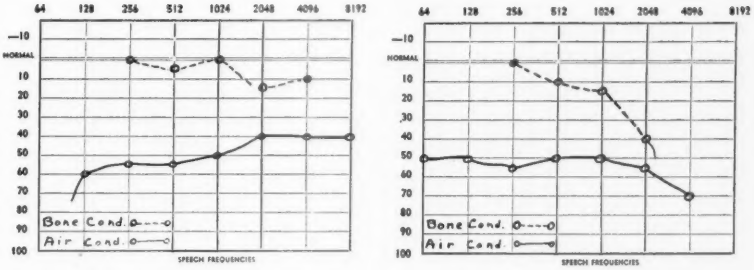

Fig. 1.—Audiogram showing air conduction and bone conduction levels of an average class A suitability case.

Fig. 2.—Audiogram showing air conduction and bone conduction levels of an average class B suitability case.

The air conduction curve may be fairly flat or have a slighly better threshold for the high than the low tones. Our Class A patients probably compare somewhat to House's[1] ideal and borderline cases combined (Fig. 1).

Class B. The bone conduction audiometric test in these cases will show the threshold for one level—usually 2048 c.p.s.—to be below 30 db. The fork tests will be similar to those in Class A except that with the 1024 fork air conduction may be equal to bone conduction and for 2048 c.p.s. the Schwabach test will usually show a slightly greater degree of impairment. The Schwabach test for the 1024 frequency may or may not be slightly shortened. The air conduction curve is usually flat or shows slightly more loss for the high than for the low tones (Fig. 2).

Class C. In these cases the audiometric bone conduction threshold is below the 30 db. level usually for two of the three speech frequencies. For the 1024 fork the Rinne test is as a rule positive. The Schwabach tests for the 1024 and 2048 frequencies are both definitely shortened. The air conduction curve almost always shows more loss for the high than for the low tones (Fig. 3).

Special attention is given to the Schwabach test for bone conduction as a check against the audiometer. We compare the patient's threshold to that of the examiner.

The general prognosis for the three classes of suitability will be discussed in connection with the end results. Since the patient is

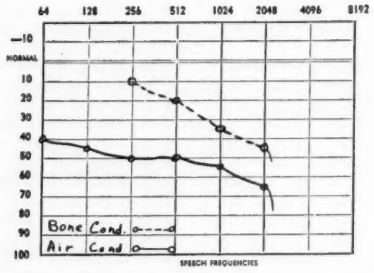

Fig. 3.—Audiogram showing air conduction and bone conduction levels of an average class C suitability case.

usually confronted with making a decision between a hearing aid and a fenestration operation the prognosis is based on the probability of the hearing improvement after surgery being equal to or better than that possible with a hearing aid.

The question of which ear is to be selected for operation will not be dwelt on in detail here. In general the poorer ear is selected for operation if cochlear function is equal in the two ears. However, if there is some difference in bone conduction levels in the two ears and an aid can be worn equally well in both ears, the ear with better cochlear function may be selected, even though it may have a slightly better air conduction level. However, we must remember that the result is always a gamble and there is about one chance in a hundred that the ear may be worse after operation than before. Other things being equal, the ear with more tinnitus should be selected for operation, inasmuch as tinnitus is partially or completely relieved in about three-fourths of patients having a fenestration.

All patients are given a complete general medical examination before operation. If there is a history of a luetic infection, this is thoroughly investigated by a competent internist to determine if sufficient treatment has been given. A history of adequately treated lues is no contraindication to this type of surgery if the patient is otherwise suitable.

End Results.—For this evaluation of over-all results two groups of cases have been studied and tabulated. The first group of 300 cases includes consecutive unselected cases for which two-year hearing tests were available. The operations on this group were done between June 1943 and July 1945. The Lempert nov-ovalis tech-

nique with the addition of continuous irrigation and a dissecting microscope was used in all these cases. A short series of stopple cases was not included in this tabulation because the results with the stopple were immediately observed to be inferior to those obtained by the regular technique. The second group is more recent and consists of 300 consecutive unselected cases which have been followed only one year. These were operated on between December 1945 and November 1946. The surgical technique in these cases was the same as that in the previous group except that a sea-sponge pack was used in Group II instead of the usual inelastic packing.[2] All preoperative and postoperative audiometric tests were made with standard audiometric technique using an interrupted tone, and going from inaudibility to audibility. The threshold then is recorded as being at the first number at which the tone is consistently heard.

There is no absolute method or standard for estimating the success of results obtained by fenestration. Particularly for patients who attain a final level of between 25 and 35 db. for the speech frequencies, the degree of success from the patient's point of view is relative. Individual factors which enter into the patient's estimation of the success of the operation are his particular needs for an excellent acuity and his degree of deafness before operation. A patient with a 65 db. preoperative level who attains a 33 to 35 db. level after operation may be better satisfied with the result than one with a 40 to 45 db. preoperative level who reaches a 28 to 30 db. level after operation.

Another factor which influences the patient's opinion of the surgical result is the difficulty he had in using a hearing aid before operation. We have observed particularly in the Class B and C cases that in those patients whose hearing improvement after operation reached only the 32 to 38 db. level, quite a few were absolutely certain that they were able to get along in their work and social contacts as well or better with that level after operation than they could with an aid before. It would seem that they were willing to sacrifice some degree of the loudness obtained by the aid, in return for being able to eliminate the distortion and interference caused by the aid amplifying background noise in certain conditions under which speech must be perceived. Of course, we must also take into consideration that the patient is anxious to be able to do without an aid and consequently may overestimate the status of his over-all hearing improvement.

In Table 1 is shown the decibel level for speech frequencies of the 193 Class A suitability cases of Group I. It will be noted that

there is a definite downward shift in some cases at the one-year level as compared to the best level. Of course, in some instances a shift of only 3 to 4 db. would change the case to the next lower group. A practical level line was not put on the chart for reasons already stated, that success obtained in fenestration surgery is in many instances a relative matter. There has been very little shift in the two-year level as compared to the one-year level. It is our plan to follow this same group of cases from year to year to see what further shift may occur in the future. While the closure factor is no longer important at the two-year period, there may occasionally be nerve changes which may become significant.

Table 2 shows a similar tabulation of the Class B suitability cases of Group I. It would seem that there is a slightly greater downward shift in these cases than in the Class A cases. Since the mechanical factors in the Class A and B cases should be essentially the same, the slight degree of nerve degeneration must account for the increased downward shift in the Class B cases. Table 3 shows the Class C cases of Group I.

In Table 4 are shown the levels of the 185 Class A suitability cases in Group II. An over-all better level has been obtained. There is a similar downward shift at the one-year period as compared to the best level. A conservative estimate would be that between 75 and 80 per cent of these patients had hearing at their one-year test which was equal to or better than that which would be obtained with a hearing aid. This is based on the statement of patients who, having obtained a level of 32 to 35 db., felt that their over-all hearing under varied conditions of environment was as satisfactory as they formerly had with an aid.[3] An audiometric study of this comparison has been reported.[4] Tables 5 and 6 show the levels of the Class B and C cases of Group II.

Table 7 shows an over-all comparison of Group I and Group II at the one-year postoperative period. Inasmuch as in Table 1 the Group I cases show little change from the one-year to the two-year test, we may be reasonably justified in assuming that the change from the one-year to the two-year period in Group II cases will be in the same proportion. While Group II shows a slightly larger percentage of patients attaining a good level of hearing, this cannot all be ascribed to improvement in technique. In comparing the preoperative levels, Group II had a better average level than Group I. Consequently we were dealing with a slightly more favorable group of cases in Group II. The first cases in Group I date back to June 1943. At that time patients with a 38 to 40 db. loss were frequently advised

to wait a year or two before having surgery, chiefly in expectation of further improvement in technique. At the present time these patients may have an operation if they desire. The result has been a slight lowering of the average preoperative level. Inasmuch as the Group II cases have a slightly better preoperative level than the Group I, less average gain is needed to bring Group II cases to a good level of hearing. Day[5] reported a series of 100 cases in which 16 patients had a preoperative hearing level ranging from 32 to 40 db. The chief reason why the Group II cases would get a better final level of hearing is that there has been less secondary nerve change. Actually, in patients who have normal cochlear function, the maximum decibel gain possible with a 40 db. preoperative level is less than that possible with a 50 db. preoperative level, even though the first patient may attain a slightly better final decibel level. For this reason Group II cases show a slightly smaller decibel average gain than do the cases in Group I.

By comparing the air conduction loss in Class A cases with that of Class B and C cases, the idea is substantiated that, if there is more than a 50 db. loss of hearing from a conduction lesion, there must be in addition some loss of nerve function. Consequently, it is unlikely that in a case in which there is an 85 or 90 db. air conduction loss it is a pure conduction deafness.

Tables 8 and 9 show the decibel gain and loss distribution. In this method of tabulation, Group I again shows a slightly better decibel gain than Group II. The reason for this has already been discussed in relation to Table 7. The most significant point illustrated by these tables is that Group II shows fewer cases with a loss than is observed in Group I. While the over-all difference in the loss columns of the two groups represented here is only about 1.5 per cent, for a given patient to be out of the loss column and possibly at a good level instead may be very important to him. Also in Group I, two year contact was lost with a few of the patients in the loss column and consequently the percentage of worse cases is actually very slightly higher than is indicated on the chart. The over-all percentage in the gains in Group I would, however, not be significantly changed by correcting this minor chance difference on the loss side. The Group II chart is entirely correct in this respect.

Of the patients operated on prior to those in Group I, there are at the present time 38 patients who have maintained a 30 db. or better level for periods varying from five to seven years.

In the early history of otosclerosis surgery, bony closure constituted the major obstacle in obtaining a permanent good result. In

our experience at the present time, complete or nearly complete physiological closure occurs relatively infrequently. The decrease in incidence of closures has been accomplished by modifying the Lempert nov-ovalis technique with the use of continuous irrigation and magnification with a dissecting microscope which makes it possible to carry out certain essential refinements in the creation of the fenestra. The details of this improved technique have recently been described by us.[2] Of the 300 cases in Group I (i.e., followed up to two years audiometrically) there were 226 patients who, at their best level test, attained a 35 db. or better level. Six of these 226 have dropped back to within 10 db. of their preoperative level. Of these 6 cases, 2 have been revised and the fenestra found to be completely open and free of scar tissue or any other visible obstruction. In one of these revised cases there was some further nerve degeneration, but not sufficient to account for the drop in hearing to the preoperative level. Of the remaining 4 cases, one patient had a cold at the time of the two-year test and in answer to a recent request for a retest, he stated he was unable to come in at the present time. He reported that as far as he knew his hearing was still at a serviceable level, and that it had improved again after his recovery from the cold at the time of the two-year test. In the remaining 3 cases we must assume that the loss of hearing occurred as a result of closure of the fenestra until some future time when a revision can be done and the issue settled one way or the other.

In those patients who have experienced a slight loss with only a partial return to the preoperative level, the reason for the drop is a matter of speculation. Some, undoubtedly, represent partial physiological closure. Others have had their drop chiefly in the higher frequencies and this may be caused by something other than a partial closure.

SUMMARY

The fenestration operation can restore a serviceable level of hearing in cases of clinical otosclerosis with suitable cochlear function and sufficient hearing loss to constitute a significant social and vocational handicap. It is our experience at the present time that in Class A suitability patients a fenestration can produce in approximately 75 to 80 per cent of the cases what may be considered a stable level of hearing which is equal to or better than that obtainable by a hearing aid. If there is some associated nerve degeneration and the case comes within our Class B suitability group, the patient has a 40 to 60 per cent chance of attaining the result mentioned

above and a Class C patient would have a 10 to 20 per cent chance. This estimate is based on the one-year level of Group II cases, having taken into consideration the fact that a small number of cases will not permanently maintain the one-year level. Class C patients and some Class B patients who wear a hearing aid well should be advised not to consider surgery. A tabulation of closures in Group I cases (followed for two years) indicates that the incidence of complete physiological closure to be 1.4 per cent in those patients who at the time of their best hearing improvement had attained a level of 35 db. or better.

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TABLE 1.

GROUP I—CASES OF "A" SUITABILITY
ON WHOM TWO-YEAR TESTS
WERE AVAILABLE

(Operated on from June 1943 to July 1945)

AVERAGE DB. LEVEL FOR 512, 1024 AND 2048	BEST LEVEL		ONE-YEAR LEVEL		TWO-YEAR LEVEL	
	NO. CASES	%	NO. CASES	%	NO. CASES	%
10—20	71	36.7	29	15.0	28	14.4
21—30	85	44.0	99	51.3	98	50.8
31—35	12	6.2	23	11.9	26	13.4
36—40	12	6.2	17	8.8	15	7.7
41—50	8	4.1	16	8.3	14	7.
51 or worse	5	2.5	9	4.6	12	6.2
Total —	193		193		193	

TABLE 2.

GROUP I—CASES OF "B" SUITABILITY
ON WHOM TWO-YEAR TESTS
WERE AVAILABLE

(Operated on from June 1943 to July 1945)

AVERAGE DB. LEVEL FOR 512, 1024 AND 2048	BEST LEVEL		ONE-YEAR LEVEL		TWO-YEAR LEVEL	
	NO. CASES	%	NO. CASES	%	NO. CASES	%
10—20	4	5.5			1	1.4
21—30	25	34.9	20	27.7	14	19.4
31—35	22	30.5	11	15.2	13	18
36—40	7	9.7	11	15.2	17	23.6
41—50	8	11.1	19	26.3	13	18.0
51 or worse	6	8.3	11	15.2	14	19.4
Total —	72		72		72	

TABLE 3.

GROUP I—CASES OF "C" SUITABILITY
ON WHOM TWO-YEAR TESTS
WERE AVAILABLE

(Operated on from June 1943 to July 1945)

AVERAGE DB. LEVEL FOR 512, 1024 AND 2048	BEST LEVEL		ONE-YEAR LEVEL		TWO-YEAR LEVEL	
	NO. CASES	%	NO. CASES	%	NO. CASES	%
10—20						
21—30	2	5.7	1	3		
31—35	6	17.4	3	8.6	2	5.7
36—40	3	8.6	3	8.6	4	11.3
41—50	12	34.2	8	22.8	8	22.8
51 or worse	12	34.2	20	57.4	21	60.
Total —	35		35		35	

TABLE 4.

GROUP II—CASES OF "A" SUITABILITY
ON WHOM ONE-YEAR TESTS
WERE AVAILABLE

(Operated on from Dec. 1945 to Nov. 1946)

AVERAGE DB. LEVEL FOR 512, 1024 AND 2048	BEST LEVEL		ONE-YEAR LEVEL	
	NO. CASES	%	NO. CASES	%
10—20	93	50.2	39	21.1
21—30	72	38.9	95	51.3
31—35	7	3.7	24	13
36—40	5	2.7	11	6
41—50	7	3.7	11	6
51 or worse	1	0.5	5	2.5
Total —	185		185	

TABLE 5.

GROUP II—CASES OF "B" SUITABILITY
ON WHOM ONE-YEAR TESTS
WERE AVAILABLE

(Operated on from Dec. 1945 to Nov. 1946)

AVERAGE DB. LEVEL FOR 512, 1024 AND 2048	BEST LEVEL NO. CASES	%	ONE-YEAR LEVEL NO. CASES	%
10—20	6	8.9		
21—30	35	52.2	24	35.8
31—35	11	16.4	16	24
36—40	7	10.4	14	22
41—50	6	8.9	11	16
51 or worse	2	3	2	3
Total —	67		67	

TABLE 6.

GROUP II—CASES OF "C" SUITABILITY
ON WHOM ONE-YEAR TESTS
WERE AVAILABLE

(Operated on from Dec. 1945 to Nov. 1946)

AVERAGE DB. LEVEL FOR 512, 1024 AND 2048	BEST LEVEL NO. CASES	%	ONE-YEAR LEVEL NO. CASES	%
10—20	1	2	1	2
21—30	9	18.7	4	8.3
31—35	7	14.5	3	6.2
36—40	12	25	14	29.1
41—50	13	27	15	31.2
51 or worse	6	12.5	11	22.6
Total	48		48	

TABLE 7.

ONE-YEAR GAIN AND LEVEL

Comparison of Group I and Group II

SUITABILITY	A GROUP I	A GROUP II	B GROUP I	B GROUP II	C GROUP I	C GROUP II
Average Preop. Level	55.3	50.7	63.4	57.9	73.2	64.2
Average Gain—All Cases	25.4	23.4	23.4	21.4	18.2	19.2
Average Gain for Cases with 30 Db. or Better Level	29.5	26.7	32.3	27.3	33.0	34.4
Number of Cases	193	185	72	67	35	48
No. of Cases with 30 Db. or Better Level	128 (66.3%)	134 (72.3%)	20 (27.7%)	24 (35.8%)	1 (3%)	5 (10.3%)
No. of Cases between 31 and 35 Db. Level	23 (11.9%)	24 (13%)	11 (15.2%)	16 (24%)	3 (8.6%)	3 (6.2%)

Note: Levels and gains are averages in decibels for 512, 1024, 2048 frequencies.

Group I—Operated on between June 1943 and July 1945.

Group II—Operated on between December 1945 and November 1946.

TABLE 8.

GROUP I—DECIBEL GAIN OR LOSS
AT ONE-YEAR TEST

SUITABILITY AVERAGE DB. GAIN OR LOSS FOR 512, 1024 AND 2048	A		B		C	
	NO. CASES	%	NO. CASES	%	NO. CASES	%
+51 to 60	1	0.5				
+41 to 50	9	4.6	7.	9.7	2	5.7
+31 to 40	58	30.0	21	29.1	5	14.2
+21 to 30	70	36.2	19	26.3	11	31.4
+11 to 20	35	18.1	15	20.8	8	22.8
+ 0 to 10	16	8.2	7	9.7	4	11.4
— 1 to 10	2	1.0	2	2.6	4	11.4
—11 to 20	1	0.5	1	1.3		
—21 to 30						
—30 to 40	1	0.5			1	2.8
TOTAL	193		72		35	

\+ Gain
— Loss

TABLE 9.

GROUP II—DECIBEL GAIN OR LOSS AT ONE-YEAR TEST

SUITABILITY AVERAGE DB. GAIN OR LOSS FOR 512, 1024 AND 2048	A		B		C	
	NO. CASES	%	NO. CASES	%	NO. CASES	%
+51 to60						
+41 to 50	3	1.6			2	4.1
+31 to 40	42	22.7	12	17.4	6	12.5
+21 to 30	69	37.3	30	44.7	13	27.0
+11 to 20	55	29.1	19	28.3	18	37.5
+ 0 to 10	14	7.5	5	7.4	5	10.4
— 1 to 10					2	4.1
—11 to 20					2	4.1
—21 to 30			1	1.4		
—31 to 40	2	1				
TOTAL	185		67		48	

\+ Gain
— Loss

XXXVII

RHINOSCLEROMA APPARENTLY CURED WITH STREPTOMYCIN

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Rhinoscleroma is a chronic granulomatous process involving the nose, upper lip, mouth and upper air passages. It is believed by many to be an infection but it is only slightly, if at all, contagious. An organism of the genus Klebsiella may usually be isolated from such patients. The chronic progressive clinical disease of rhinoscleroma has not been reproduced in animals but the organisms are always present in the tissue of patients with this clinical manifestation. The organisms are usually seen in the large phagocytes and are easily grown in an artificial culture medium. Organisms with similar cultural features are frequently isolated from the respiratory tract of normal persons.

The disease was once thought to exist only in Central and Southeastern Europe but it now occurs anywhere and the incidence seems to be increasing in South and Central America. According to Cunning and Guerry,[1] a total of 102 cases had been reported in the United States and Canada by 1942; 16 of the patients were native-born.

Clinically, there is first an insidious growth of hard smooth nodules in the anterior nares. This growth spreads gradually until, in many cases, protruding, rounded, smooth masses with intact skin surface close the nostrils. From the anterior part of the nose the lesion spreads backward into the pharynx, larynx and trachea and even into the bronchi. It may extend forward onto the upper lip or involve the cheeks, gums, antrum, lacrimal ducts, soft and hard palates and tonsils. Because of the presence of these masses function is impaired, and such symptoms as foul nasal discharge, nasal ob-

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struction, deafness, tinnitus, hoarseness and difficult breathing may ensue. Tracheotomy is often necessary. The disease is limited to the mucous membrane and skin, ulceration is the exception and not the rule, and constitutional symptoms are rare.

Up to the present no satisfactory method of treatment has been available. Therapy with roentgen rays or radium is, at best, palliative and must be repeated as recurrence of the disease dictates. Sulfanilamide and its derivatives seem to control the secondary infection but they have no effect on the scleromatous tissue.

Recently a patient with rhinoscleroma has been treated at the clinic with streptomycin.

REPORT OF A CASE

A white woman, aged 32, of Russian descent, born in Siberia and reared in the Far East, was first seen at the clinic on September 4, 1946. Her chief complaint was bilateral nasal obstruction for 15 years with hoarseness, a sense of constriction in her throat and slight difficulty in swallowing for one year. A diagnosis of rhinoscleroma had been made in 1938 in Shanghai, China, where she lived. Treatment with autogenous vaccine apparently arrested the disease process except for a short period in 1941 when there was reactivation. In 1945 the case was reported[2] as one of rhinoscleroma which had been arrested by autogenous vaccine.

On examination at the clinic both nostrils were found to be almost completely obstructed with granulomatous tissue. There was scarring of the pharynx, with a small opening into the nasopharynx. The posterior surface of the epiglottis and the left side of the larynx were involved with granulomatous masses. Tomographs of the larynx showed almost complete obstruction of the airway at the level of the glottis. This was so marked that tracheotomy followed by skin grafting of the larynx was considered.

With the aid of suspension laryngoscopy, biopsy specimens were taken from the larynx on September 13, 1946. On microscopic examination a diagnosis of chronic granuloma, probably rhinoscleroma, was made. Culture of the granulomatous material showed large numbers of Klebsiella.

Previous workers[3-6] have shown that organisms of the Klebsiella genus are sensitive to streptomycin. It was expected, therefore, that if the Klebsiella strains commonly found in such clinical conditions were etiologically related to the disease process, strepto-

mycin might have therapeutic value. For these reasons the organism isolated from this patient was tested for sensitivity to the drug. It was found that growth was inhibited by 0.3 unit per milliliter of medium. Accordingly on September 27, 1946, treatment with streptomycin was begun. The patient received 250 mg. of streptomycin intramuscularly every three hours. After the first week, that is, after 12.75 gm. of streptomycin had been administered, the antibiotic was discontinued because of joint pains, a cutaneous rash, malaise and fever. After twelve days' rest it was possible to continue the administration of streptomycin for 43 consecutive days or until a total of 97.25 gm. had been given. Treatment with the antibiotic was discontinued on November 26, 1946, because of dizziness and slight nausea.

The patient did amazingly well. After 18 gm. of streptomycin had been given, she stated that she felt 50 per cent better than before treatment, that the condition of her nose was better and that the laryngeal obstruction was less obvious.

After 38 gm. of streptomycin had been administered, the patient stated that there was only one third as much mucopurulent material and crusting in her nose as there had been before treatment. After 53 gm. had been used, tomographs of the larynx showed a good airway, and suspension examination of the larynx showed some scarring anteriorly and on the left side but no evidence of active disease was present. The results of culture of tissue removed from the nostrils were negative for Klebsiella. After 95 gm. of streptomycin had been given, again the results of culture of tissue from both nostrils were negative for Klebsiella and clinically the patient was cured.

The patient suffered from varied toxic manifestations ascribable to the streptomycin during the course of treatment. The dermatitis, fever and arthralgia subsided rapidly after administration of streptomycin was discontinued and did not recur when treatment with the antibiotic agent was resumed. Just before the completion of treatment, symptoms of reduction in the vestibular function of the eighth cranial nerve developed, as manifested by a disturbance in equilibrium and slight nausea. The nausea subsided soon after the use of streptomycin was stopped. The disturbance in equilibrium persisted for several weeks but the patient was able to compensate for this reduction in vestibular function. At the time of the patient's dismissal, there was no apparent disturbance in equilibrium.

On March 19, 1947, approximately four months after the administration of streptomycin had been discontinued, suspension ex-

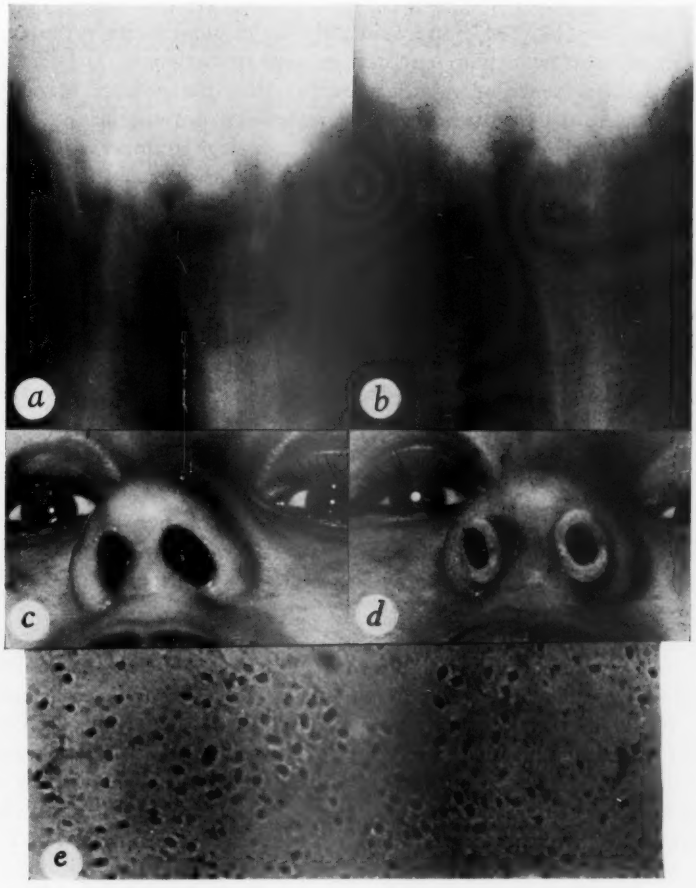

Fig. 1—*a*. Tomograph made before treatment, September 4, 1946. Note the small airway at the glottis. *b*. After treatment, November 1, 1946. Note the increase in the size of the airway. *c*. Nostrils after treatment. The skin graft can be seen in the right nostril. *d*. The vernonite obturators are in place. These should be worn from time to time, possibly for six months, in order to maintain the airway. *e*. Klebsiella organisms.

amination of the larynx revealed no activity and again the results of culture of material from the nostrils were negative for Klebsiella.

On July 14, 1947, approximately eight months after the cessation of treatment with streptomycin, examination of the larynx showed no evidence of the disease other than the residual scarring, and the results of culture of tissue removed from the larynx were negative for Klebsiella. The presence of large numbers of these organisms in the tissue before treatment and their rapid disappearance, with clinical recovery, under treatment with streptomycin suggest that they may have been causally related, at least in part, to the pathologic process.

On July 25, 1947, the cicatricial stenosis of both nostrils was corrected by dissecting out the scar tissue produced by the disease and relining the nostrils with shaved grafts placed over soft rubber molds. The grafts were taken from the abdomen. Later, perforated vernonite obturators replaced the molds in each nostril and the patient was advised that they should be worn from time to time, possibly for six months.

The patient was dismissed from our care with no evidence of active rhinoscleroma, September 16, 1947, approximately ten months after treatment with streptomycin had been completed (Fig. 1).

This patient with rhinoscleroma is the first to be reported as probably cured with streptomycin, as far as we know. At the time of the original examination, since the airway was partially obstructed, it was thought that tracheotomy would be necessary; as much of the granulomatous and scar tissue in the larynx and trachea as possible would then be removed and replaced with a skin graft and later with an obturator in order to maintain an opening. The use of streptomycin cleared up the granulomas and also eliminated Klebsiella from tissue taken subsequently for biopsy. This evidence adds to the general impression that Klebsiella is an etiologic factor in the disease. The tomographs show very strikingly the increase in the diameter of the airway following the treatment.

MAYO CLINIC.

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XXXVIII

THE EFFECTS OF NEGATIVE AIR PRESSURE IN THE MIDDLE EAR

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An earlier study[1] dealt with the effects upon the ear of positive air pressure introduced into the tympanic cavity. The present experiments deal similarly with negative pressure. The situation is like that encountered when we descend rapidly from a high altitude to a low one and do not equalize the pressure in the middle ear with that of the atmosphere.

These experiments, like the foregoing, were carried out in cats and made use of the electrical potentials of the cochlea to show how the ear's efficiency in sound reception is affected by the pressure changes.

The general procedure follows that already described in our earlier paper. Here, however, we have made use of a mediolateral approach to the bulla instead of the usual ventral approach. This approach takes only a little more time than the other and provides a flatter field. The final stage of the operation followed the procedure formerly worked out. Two holes were opened in the bulla, one about 2 mm. in diameter near the jugular process of the occipital bone, and the other about 5 mm. in diameter farther toward the mesial side. Both holes were reamed and fitted with rubber stoppers. Through the smaller stopper ran the electrode wire with a tip of platinum foil, and by looking through the other hole (with its stopper removed) this tip was brought into contact with the round window membrane. The larger hole then was closed with a stopper through which a glass tube was inserted, and this tube was connected by rubber tubing to the negative pressure apparatus. An

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This research was carried out under contract with the Office of Naval Research, as Contract N6 onr-270, Task Order III.

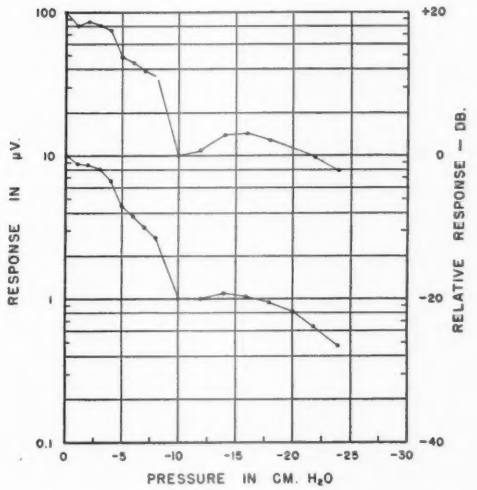

Fig. 1.—The effects of negative pressure on the responses to a 5000 cycle tone, presented at two different intensities.

indifferent electrode was located in the masseter muscle. When necessary, the eustachian tube was kept closed by pressing on its membranous portion with a spade-shaped prod.

Negative pressure was produced with a large hypodermic syringe and measured with one of three gauges covering different ranges from 1 mm. of water to 300 mm. of mercury.

The animals were anesthetized with nembutal injected intraperitoneally. In most of the experiments, a purified form of curare (Squibb's Introcostrin) was injected into the femoral vein to suppress the action of the tympanic muscles, and the animal then was maintained by artificial respiration.

Pure tone stimuli were delivered from a loudspeaker to a tube at the animal's ear, and the resulting cochlear potentials recorded with a General Radio Type 736-A wave analyzer used as a voltmeter. The tests were carried out on 14 ears.

RESULTS

A reduction in the air pressure of the middle ear cavity causes usually a marked drop in the cochlear potentials. Fig. 1 shows

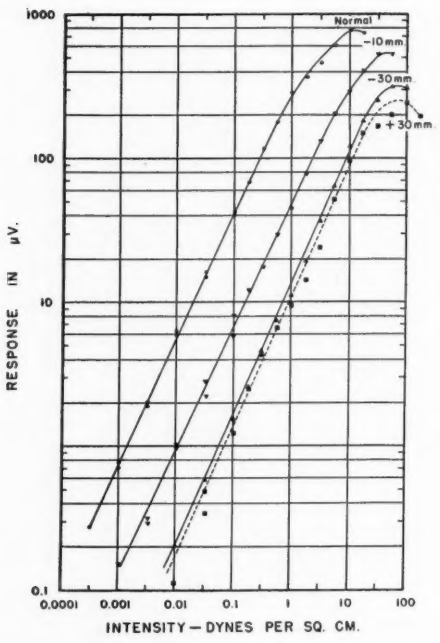

Fig. 2.—Intensity functions for a tone of 1000 cycles, under normal conditions and under pressures of −10, −30 and +30 mm. of mercury.

typical results for a tone of 5000 cycles when presented at two different intensities. The tone was first adjusted in intensity to give a response of 100 microvolts for the normal condition of zero pressure, and with the intensity maintained steadily at this level the air pressure was reduced by successive steps until a value of −25 cm. of water was reached. The cochlear potentials fell away as shown by the upper curve. Then the air pressure was restored to normal and the stimulus intensity adjusted downward to give a response of 10 microvolts and held at this value. Negative air pressures were introduced as before and a second series of potential measurements obtained, as shown in the lower curve.

The two curves are strikingly similar, and such correspondence is the rule so long as moderate intensities are used. Fig. 2 shows these relations more extensively, in the form of intensity functions. The function to the left represents the normal variations in the coch-

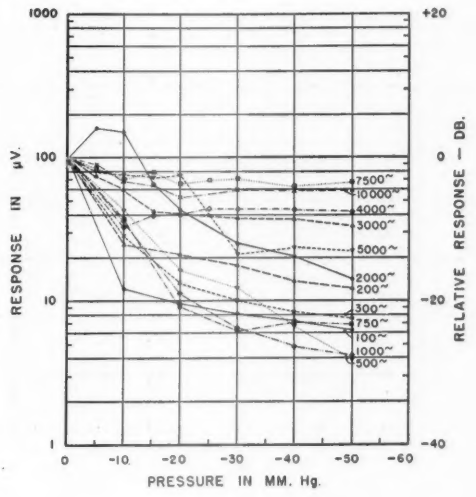

Fig. 3.—Negative pressure curves for various tones, whose frequencies are shown at the right ends of the curves.

lear potential obtained with a tone of 1000 cycles presented at various intensities. Then the air pressure was reduced to −10 mm. of mercury and the intensity varied to obtain a second function, as shown in the next curve to the right. The same was done for an air pressure of −30 mm. Finally, for comparison, the pressure was raised to 30 mm. above zero and a function obtained as shown. These functions are simple in form and depart but little from linearity except at high levels where overloading enters.

The impairment of sensitivity that the first figure showed as a drop in the curve here is represented as a shifting of the function to the right, without any notable alteration of form outside the region of overloading. In this region, however, a variation appears; in the presence of pressure, both negative and positive, the bending occurs at a lower level of response than it does normally. This feature appears in some ears and not in others and its possible cause will be discussed later.

In Fig. 3 are shown pressure curves for a large number of frequencies. They are of much the same form. The curve for 2000 cycles is exceptional in undergoing a preliminary rise, after which it suffers a fall like the others. The amount of the impairment

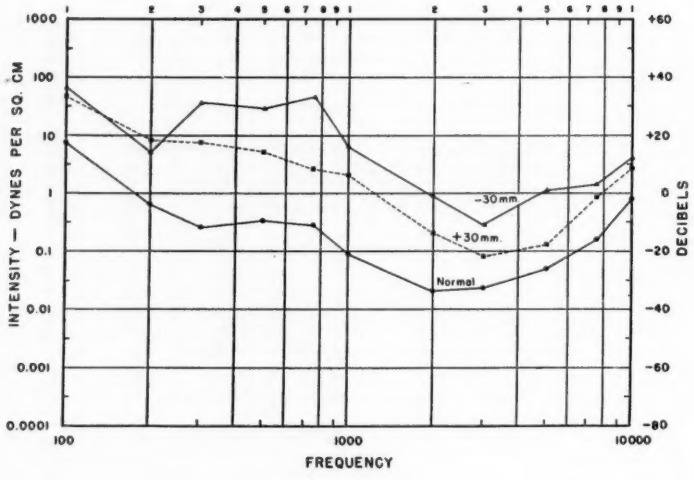

Fig. 4.—Equal-response curves for the normal condition and for pressures of −30 and +30 mm. of mercury.

caused by the pressure change varies with frequency: the highest tones are affected relatively little.

Figure 4 shows in another fashion the relation to frequency and the sensitivity function of the ear. These are equal-response curves: each curve represents, over the frequency range shown, the stimulus intensity required to produce a standard potential of 10 microvolts. The lowermost curve is for the normal condition, the uppermost curve for a negative pressure of 30 mm. of mercury, and the curve between for an equal positive pressure. In this instance the effect was somewhat greater for negative than for positive pressure; in some others little difference was found. The impairment of sensitivity, which is shown here as a raising of the curve, is greatest in the medium low frequencies and progressively less as the frequency is raised.

There are effects of negative pressure also when the ear is stimulated by bone conduction. For these measurements the tones were applied with a crystal vibrator that actuated a bakelite rod pressed firmly against the lateral prominence of the mastoid bone. Fig. 5 gives some of the results. Each tone was adjusted in intensity to give 10 microvolts of response under the normal condition, and then

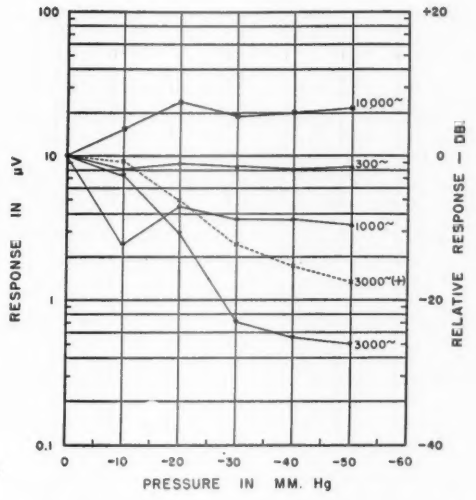

Fig. 5.—Pressure curves for bone-conduction stimulation.

the air pressure was reduced by steps of 10 mm. of mercury until a value of −50 mm. was reached. The results varied according to the frequency, as shown. For 300 cycles the effect was slight. For 1000 cycles there was a rather marked drop at first, then a rise, and finally a levelling off. For 3000 cycles the effect was large, even larger than was found for air conduction. For 10,000 cycles the effect was an augmentation of response. The broken curve in this figure represents the effects of positive pressure at 3000 cycles; for this curve the abscissa scale must be taken as positive instead of negative. It is evident, on comparison of these curves with those of Fig. 3, that negative pressure operates differently on air- and bone-conducted sounds.

In three of the experiments the strength of the eardrum was tested by carrying the pressure reduction to the point where the drum was ruptured. This point was −89 mm. of mercury in one ear and −140 mm. in the two others.

DISCUSSION

While some data on positive pressures have been given here for comparison, a reference to our earlier study will permit a more thorough-going examination of the effects of these two kinds of

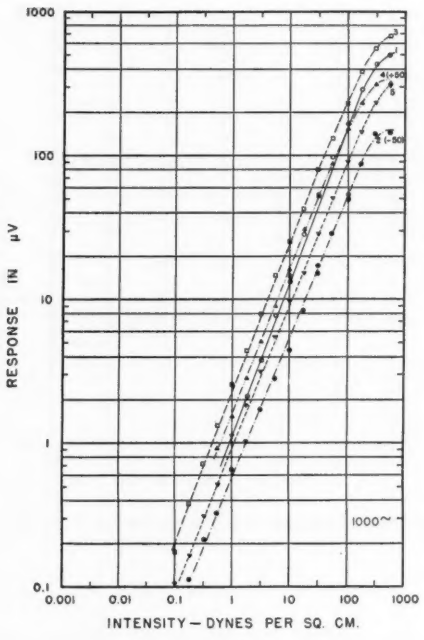

Fig. 6.—Intensity functions in a pathological ear. The functions are numbered in order of the observations; Nos. 1, 3, and 5 are under zero pressure, No. 2 under −50 mm. of mercury, and No. 4 under +50 mm. of mercury pressure.

pressure change. In general, the similarities are great. The forms of the curves as pressure varies are much the same. For both negative and positive changes the low tones are affected more than the high tones. Also for both there is a narrow region of frequency, around 2000 cycles, where the smaller changes give an augmentation of response. There is also a correspondence in the effects obtained by bone conduction. The curves are very different from the air-conduction curves, and for the most part the changes are smaller in amount. For both kinds of pressure the bone-conduction responses to the highest tones are affected relatively little.

The results on positive pressures were ascribed principally to action on the conductive system of the ear, especially the eardrum. The same explanation serves for negative pressures. Either kind of pressure displaces the drum from its normal position and in so doing

adds both stiffness and damping to this mechanism. The increase of stiffness explains the variation of the effect with frequency; a tense membrane, in the range below its resonance point, will favor the transmission of the higher tones.

The increase of stiffness will raise somewhat the resonance frequency of the conductive system. No doubt this shift in resonance accounts for the augmentation of response shown for certain tones on the introduction of pressure changes. The augmentation is found in the region of greatest sensitivity of the ear as shown by the cochlear potentials. Theoretical considerations will lead us to suppose that it obtains for frequencies just above the normal resonance point. When pressure is applied to make the drum more tense, the resonance point is shifted upward to these frequencies. As the pressure change is made greater, however, the damping effect grows relatively rapidly, and the response falls away.

There are also inner ear effects for the two kinds of pressure variation. These effects enter especially into the changes observed for bone-conducted stimuli. It must be added at this point, however, that bone conduction is affected by the middle ear mechanism when it is present, and the effects shown are not to be taken as due exclusively to action on the inner ear.

The reduction of the maximum value obtained for the cochlear response, as shown in Fig. 2, was observed in some ears but not in others. In this instance the reduction amounted to 8.5 db., while in another ear it reached the rather formidable value of 20 db. It is not due to activity of the tympanic muscles because it is found in well-curarized animals as well as in those under ordinary anesthesia. It appears most noticeably when the animal is not very sensitive to begin with, and after pressure is applied, a very high level of stimulation is necessary to delineate the upper portion of the function.

We suggest two possible causes of this reduction. We know from the bone-conduction studies, especially those in which the ossicular chain is broken, that the pressure affects the inner ear. It may do so in such a manner as to curtail the processes through which the cochlear potentials arise.

A second possibility is middle ear distortion. While ordinarily this kind of distortion is negligible in relation to the inner ear distortion[2, 3] it is not so always. Its presence has already been demonstrated when heavy tension is placed on the stapedius tendon.[4] Now it appears likely that in certain ears under pressure, when strong

stimuli are tolerated by the inner ear because the transmission is reduced, this kind of distortion can make itself felt. This possibility needs to be explored further.

The usual effects of pressure are much modified in ears in which there is some pathological condition that impairs the mobility of the drum. When the drum is thickened and scarred or blocked by an accumulation of foreign substances such as inflammatory secretions and cerumen, the application of pressure may affect the responses but little, or may alter the general condition of sensitivity in ways difficult to predict.

Figure 6 shows the effects of pressure in an ear in which a massive plug of wax filled the deeper portion of the external auditory meatus and made close contact with the drum. Five functions are given, numbered in the order in which the measurements were made. Curve 1 shows the initial condition, before any pressure changes were made. Then a negative pressure of −50 mm. of mercury was introduced and maintained for two or three minutes during which the readings of Curve 2 were obtained. The responses were reduced about 10 db. as shown, which is considerably less than usually found for this degree of pressure change. Then the pressure was restored to normal and another set of readings obtained, as shown in Curve 3. The condition of the ear had been changed as a result of the maintenance of negative pressure; the responses were enhanced by about 4 db. Evidently the drum was drawn away from the wax plug and its mobility improved. Thereupon a positive pressure of 50 mm. of mercury was applied and Curve 4 obtained. This curve represents only a slight reduction from the normal function just obtained which again is unexpected for this degree of pressure. Finally this pressure was removed and a third normal function obtained, as shown in Curve 5. This function represents a reduction of about 9 db. from the normal function just preceding, and a reduction of 4 db. from the initial condition. We can attribute it to the action of the pressure in forcing the drum into firm contact with the wax plug and immobilizing it further.

These results on the effects of pressure in both normal and abnormal ears are of interest in relation to certain clinical observations in man. Oftentimes when an inflammation involves the upper region of the pharynx, the eustachian tube fails to open as it does ordinarily during swallowing, and the usual ventilation of the middle ear cavity is absent. Then by absorption of this air a negative pressure is produced in the middle ear cavity and the drum becomes retracted. Besides subjective symptoms of fullness and discomfort

there is usually a noticeable impairment of hearing and sometimes an irritating distortion of sounds as well.

Treatments devised for this condition involve different ways of introducing air into the middle ear cavity by way of the eustachian tube. Such treatments may produce an improvement of hearing, though for the most part only a temporary one. Politzer,[5] who did much to popularize this form of treatment, explained its effects as due to the restoration of the drum to its normal position and a removal of undue tensions in the ossicular chain.

We remark again on the curious instability of an ear exposed to pressure changes. What was described earlier for positive pressures was found here for negative pressures likewise: after any change of pressure a period of slow adjustment usually ensues, during which the potentials only gradually settle down to an equilibrium value. Typically, when a positive or negative pressure is introduced the response falls abruptly at first, rises rapidly for a time, and then makes a slow approach to a final value. In some instances the rise overshoots the final value and settles down to it, or even waveringly crosses it several times before settling down. The amount and the rate of this equilibration process varies greatly in different animals; often it takes as long as two minutes. It is certain that this instability is not an activity of the middle ear muscles, for it occurs in curarized animals and is seen in the responses that remain for a time after death. We have supposed it to be mechanical and to lie in the middle ear system; but it is also possible that it has its origin in the inner ear processes.

SUMMARY

A decrease in the air pressure of the middle ear cavity causes usually a reduction in the electrical responses of the cochlea. The only exception to this rule is found for certain tones in the region of highest sensitivity, which are slightly improved by the smaller pressure changes; but these tones too suffer impairment when the pressure changes are large. In general, the low tones are affected more than the high tones.

The effects of negative pressures follow closely those already disclosed for positive pressures. The sensitivity is reduced, but the form of the intensity function over the greater part of its course is essentially unaltered. Effects occur both for air-conducted and bone-conducted sounds, but their amounts differ greatly.

The evidence indicates that the pressure operates mainly upon the transmission mechanism, especially the eardrum, and its action is to increase both the stiffness and the damping of these parts. Some effect also is referred to the inner ear.

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XXXIX

CARCINOMA OF THE TRACHEA

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Any instance of carcinoma of the trachea seems worthy of report in view of the rarity of the condition. This is all the more true now that better roentgenographic methods and bronchoscopy are generally available, for cancer of the trachea is no longer merely of academic interest as a pathologic curiosity but is one of the conditions of the upper respiratory tract that the practicing physician may be called upon to recognize and treat. Since the condition is so rarely encountered, only the collective experience of the entire profession can provide sufficient basis for formulating any conclusions that may add to our knowledge of this type of lesion.

The first case I have to present is definitely one of primary carcinoma of the trachea. The patient was first observed two years ago and was treated by roentgen irradiation. Recently he has had a recurrence of symptoms and of the carcinoma and is now undergoing another course of radiation therapy. In the second case, observed by my associate, Dr. S. C. Missal, the carcinoma was so far advanced when the patient was first observed that it could never be definitely determined, even at autopsy, that the lesion was primary in the trachea.

CASE 1.—A man, aged 47 years, was admitted to St. Vincent Charity Hospital for treatment of an illness which had begun eight weeks before with incessant tickling in the throat and continuous coughing. The patient complained also of shortness of breath and wheezing which had become progressively more severe. During the last week, the cough had been productive of small amounts of blood and frothy sputum.

Fluoroscopic and radiographic examination (Figs. 1 and 2) of the chest showed the presence of a sharply circumscribed spherical tumor, about 6 cm. in diameter, in the upper mediastinum just to the right and behind the ascending arch of the aorta, lying in intimate relationship with the esophagus, which it compressed, and with

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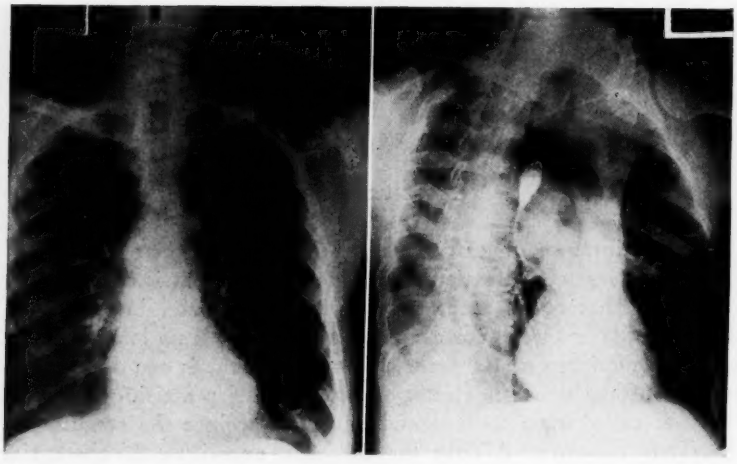

Fig. 1, Case 1.—April 23, 1946—Posterior-anterior view of the chest showing a sharply circumscribed spherical tumor approximately 6 cm. in diameter in the upper mediastinum and to the right of the arch of the aorta.

Fig. 2, Case 1.—April 23, 1946—Left oblique view of the chest showing the upper mediastinal mass lying behind the arch of the aorta and compressing the esophagus.

the trachea at its bifurcation. The tumor showed no calcification and there was no similar tumor elsewhere. The lungs appeared clear and well aerated, indicating absence of bronchial obstruction. The heart and the great vessels appeared normal in size and shape. The roentgenologic diagnosis was right upper mediastinal tumor of undetermined origin.

Tracheoscopy disclosed a large fungating mass extending over the posterior wall of the trachea, most prominent at 5 o'clock. This was situated 25 cm. from the gum line, just at the bifurcation. The tumor bled freely and was quite friable. Esophagoscopic examination showed nothing abnormal.

Histologic examination of tissue removed from the trachea showed that it was composed of necrotic tissue and fibrinopurulent exudate with associated tumor. The tumor was characterized by compact masses of moderately pleomorphic neoplastic epithelial cells displaying no architectural arrangement and no evidence of keratin-

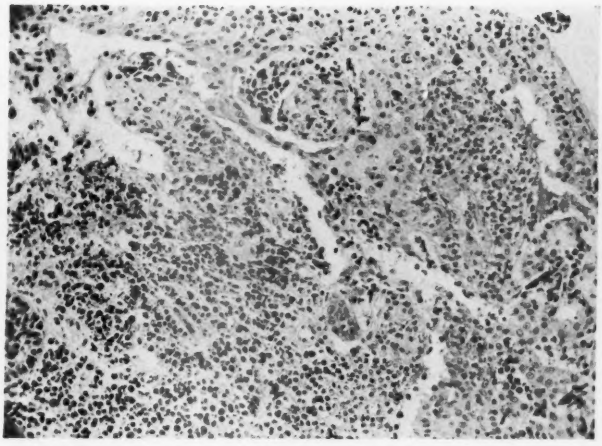

Fig. 3, Case 1.—Photomicrograph of section of the tracheal tumor with necrotic tissue and fibrinopurulent exudate. The tumor was characterized by compact masses of moderately pleomorphic neoplastic epithelial cells displaying no architectural arrangement and no evidence of keratinization, with numerous mitotic figures.

ization. Numerous mitotic figures were evident. The pathologic diagnosis was poorly diffentiated squamous carcinoma (Fig. 3).

Roentgen therapy was instituted immediately, and the patient was discharged from the hospital five days after admission, to continue roentgen treatments as an out-patient. A week after his discharge, he became dyspneic, cyanotic and apprehensive, and reentered the hospital in acute respiratory distress. His temperature at that time was 37.8° C.; pulse, 120; respirations, 30, and the blood pressure was 160 systolic, 100 diastolic. Examination of the chest showed dullness at the base of the left lung and decreased breath sounds, indicating atelectasis. The right lung was hyperresonant.

Since I was out of town at the time this episode occurred, the patient was cared for by Dr. Missal. Because the tumor had demonstrated a tendency to bleed excessively, he wisely decided against performing a bronchoscopy; instead he passed a catheter and instilled adrenalin and pontocaine to reduce the edema and loosen the secretions. Oxygen was administered through the tube. After this procedure, the patient coughed up a considerable quantity of thick, blood-tinged sputum. When the tube was removed, breathing im-

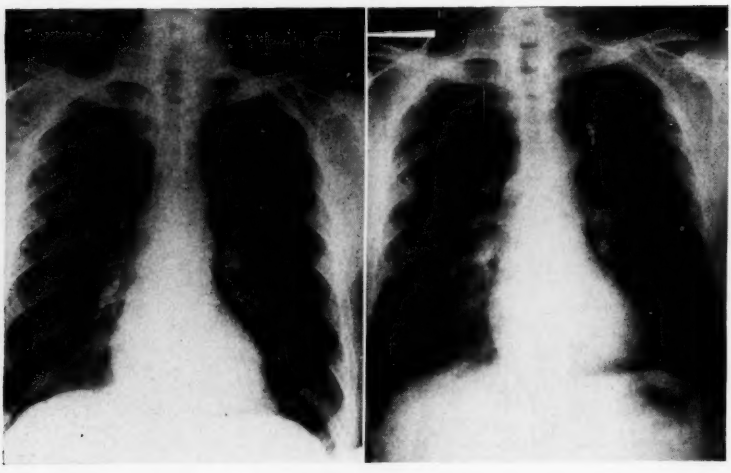

Fig. 4, Case 1.—May 28, 1946—Posterior-anterior view of the chest showing no evidence of tumor.

Fig. 5, Case 1.—February 13, 1948—Posterior-anterior view of the chest showing no change in the anterior mediastinal shadows but apparently some widening of the mediastinum at the level of the seventh and eighth ribs posteriorly.

proved and the left lung showed evidence of re-expansion. By the following day, a roentgenogram showed that both lungs were aerated equally and fully. After ten days, the patient was dismissed from the hospital.

The course of roentgen irradiation was administered in 34 days. This was given through six fields, each 6 to 8 cm. in diameter, with the central ray directed toward the tumor by fluoroscopic localization. Two fields were irradiated daily through a filter of 0.5 mm. of copper. Other factors were 200 kv, 1.0 mm. copper half-value layer, 70 cm. distance. The posterior field received 2800 r (in air) in 25 days and each of two anterior and posterior oblique fields received 2050 r (in air) in 20 days. The estimated quantity of radiation received by the tumor was 4300 r.

Roentgen treatment was completed on June 3, 1946. On May 28 a roentgenogram of the chest showed no evidence of the lesion (Fig. 4). The patient experienced complete relief of symptoms and gained 15 pounds in weight during the course of radiation treatment.

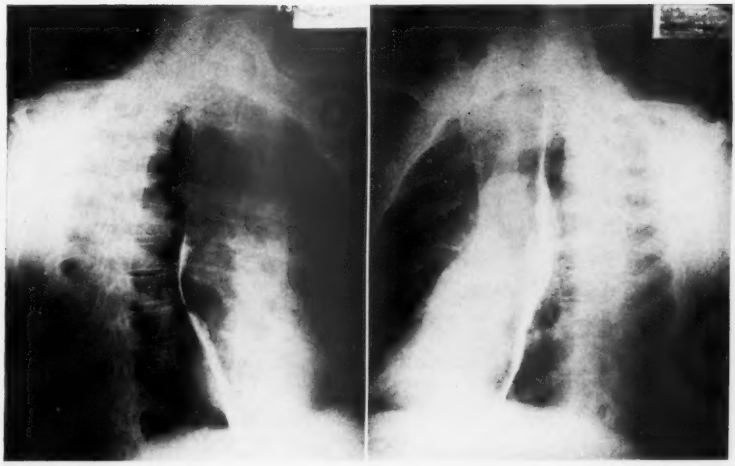

Fig. 6, Case 1.—February 17, 1948—Left oblique view of the chest showing slight depression of the esophagus in the posterior mediastinal region, but no distinct soft tissue mass is identifiable.

Fig. 7, Case 1.—February 17, 1948—Right oblique view of the chest showing no evidence of tumor.

Within the next few weeks, he gained an additional seven pounds. Repeated fluoroscopic examinations on June 28, October 15 and December 27, 1946, and on April 16, 1947, were all negative for evidence of tumor, and the patient remained in excellent health generally.

On February 13, 1948, the patient reported that he had had a recurrence of coughing and wheezing during the last week. He stated that his general health had been good and the weight gain which had followed radiation treatment two years before had been maintained.

An anterior-posterior view of the chest showed that the anterior mediastinal shadows had not changed since the last examination, but there appeared to be slight widening of the mediastinum at the level of the seventh and eighth ribs posteriorly (Fig. 5). A left oblique view showed a slight depression of the esophagus in the posterior mediastinal region, but no distinct soft-tissue mass could be identified (Fig. 6). A right oblique view (Fig. 7) also showed no evidence of a tumor.

Bronchoscopy was performed on March 5, 1948. A mass was encountered just below the subglottic space on the anterior wall of the trachea. This measured 1.5 cm. in diameter. A fungating mass was also present at the site of the original lesion. Vision into the right main bronchus was not obstructed, but the bifurcation was not evident and the orifice of the left main bronchus could not be seen. The tumor in the lower part of the trachea extended from the posterior wall upward laterally and seemed to be extending also into the left main bronchus. Biopsy specimens from both tracheal lesions were removed for histologic study. The microscopic picture was identical in both and the same as that of the original growth.

The patient is now receiving a second course of roentgen treatment. It is planned to administer the radiation at 50 cm. distance, through two portals (15 x 15 cm.) to the lesion at the lower end of the trachea. Both anterior and posterior fields will receive 1500 to 2000 r (in air) according to the patient's tolerance. The plan of treatment of the upper tracheal lesion is to give 2000 to 3000 r (in air) anteriorly and posteriorly through a 4-cm. field at 50 cm. distance.

Comment.—This patient's case history focuses attention on several facts that are interesting from the clinical standpoint. He was examined elsewhere a few days before I saw him and was told that a chest roentgenogram showed nothing abnormal. Furthermore, although his symptoms of dyspnea, wheezing and hemoptysis clearly indicated the need for a bronchoscopy, this was not done, apparently because the negative chest roentgenogram allayed any suspicion the examining physician may have had as to the seriousness of the condition.

It has been amply demonstrated that special radiologic methods must be used to reveal tumors of this type; the routine anterior-posterior roentgenogram of the thorax is not reliable for this purpose. Oblique and lateral views and use of a contrast medium in the esophagus and fluoroscopic examinations with the patient changing positions, coughing, and swallowing may be necessary. This has been stressed by Brown, Weiss, Iglauer and Fine,[1] Tinney, Moersch and McDonald,[2] Rathbone[3] and Boemke and Moritz.[4] Rathbone's patient had had repeated roentgenologic examinations of the neck and chest over a five-year period which had been reported negative, while the dyspnea of which she complained became progressively more severe. The tumor was finally discovered radiologically by asking the patient to swallow while in the lateral position. In the case reported by Boemke and Moritz, the patient also had had symptoms for five

years and repeated radiologic examinations of the chest revealed no evidence of tumor. Guttman[5] and Penta[6] have also reported cases in which patients with tracheal tumors had been undergoing treatment for asthma while their dyspnea and wheezing progressed to serious respiratory distress. These occurrences make it evident that despite all that has been said and written to show that bronchoscopy is indicated in most cases of respiratory difficulty, there is still need for reiteration and emphasis of this fact to the medical profession generally.

Although the present outlook in this case is certainly not good, the history so far would seem to indicate that it may not be entirely hopeless. This patient originally had a highly malignant lesion on the lower posterior wall of the trachea, yet his response to irradiation with roentgen rays enabled him to enjoy good health and to carry on his work and other normal activities for two years before there were any signs of recurrence. The prognosis must necessarily be guarded at this time, but there are a few reports in the recent literature which give some basis for the hope that radiation treatment of the recurrent lesion may also be productive of considerable benefit.

Brown, Weiss, Iglauer and Fine[1] reported a case in which the patient remained symptom free for three years after roentgen treatment for a recurrence of squamous-cell carcinoma of the trachea. Orso[7] reported a secondary squamous-cell carcinoma of the trachea which occurred six months following an operation for a similar cancer of the larynx. After removal through the bronchoscope, the tracheal tumor was treated by roentgen irradiation and ten years later the patient had had no recurrence of the lesion. A patient with primary squamous-cell carcinoma of the trachea, reported on by Olsen[8] and also by Tinney, Moersch and McDonald,[2] was known to be alive nine years after treatment with diathermy and radium.

Numerous other cases of tracheal tumors—probably of less malignant type than these squamous-cell carcinomas, although in some there were recurrences—in which the patients survived one to several years following some form of radiation therapy have been reported by Cann,[9] Engelstad,[10] Pierson,[11] Fisher,[12] Dean[13] and others.[2, 6] The evidence seems to indicate that all the patients whose treatment for tracheal tumor has been of significant benefit have received either roentgen or radium irradiation. In some of these cases, surgical removal by open operation or through the bronchoscope was also carried out.

Another interesting feature of the present case is that in addition to the recurrent tumor at the original site on the lower posterior wall there is also another tumor, of identical structure, higher in the trachea and on the anterior wall. This tumor is apparently entirely distinct from the original lesion which seems to be extending downward into the bronchus rather than upward. The question arises as to just how the new tumor should be classified. Is it an extension, a metastatic lesion or a new primary tumor? This occurrence is somewhat comparable to that in the case reported by Orso,[7] who believed the tracheal lesion was an implant from the previous laryngeal operation. Watson[14] also reported on a patient with tracheal cancer who had had a laryngofissure and radium treatment 15 years previously for a squamous-cell carcinoma of the larynx. The tracheal cancer developed on the posterior wall of the trachea where the tracheotomy tube came in contact with the mucosa and was removed by an open operation. Watson was inclined to believe that this lesion was not directly related to the laryngeal cancer but that it was another primary growth resulting from chronic irritation of the mucosa by the tracheal tube.

CASE 2.—A woman, aged 66 years, was admitted to the accident room of St. Vincent Charity Hospital on June 14, 1947, because of extreme respiratory distress. She was sent immediately to the operating room for a tracheotomy.

A midline incision was made and a portion of the third tracheal ring was removed. A No. 6 tracheotomy tube was inserted but could not be passed because of a tracheal obstruction. The tracheotomy tube was removed and a 7-mm. bronchoscope revealed a tumor mass almost totally occluding the trachea. A smaller bronchoscope was then passed beyond this mass, and the patient began to breathe more easily. With the bronchoscope in place, the incision was opened downward, exposing a large portion of the trachea. The tumor extended too far downward to make its total removal possible, however. Pieces of it were excised for histologic study and a long-stem No. 5 tracheotomy tube was inserted. After this procedure, the patient was able to breathe with less difficulty.

A posterior-anterior roentgenogram of the chest showed the tracheotomy tube in place and revealed a pronounced widening of the upper mediastinum (Fig. 8). There was no evidence of metastatic or parenchymatous lesions of the lungs. The heart shadow was of normal size, but it apparently was displaced somewhat to the left, possibly owing to scoliosis of the middorsal spine or to the rotational position of the patient. A lateral view showed that the

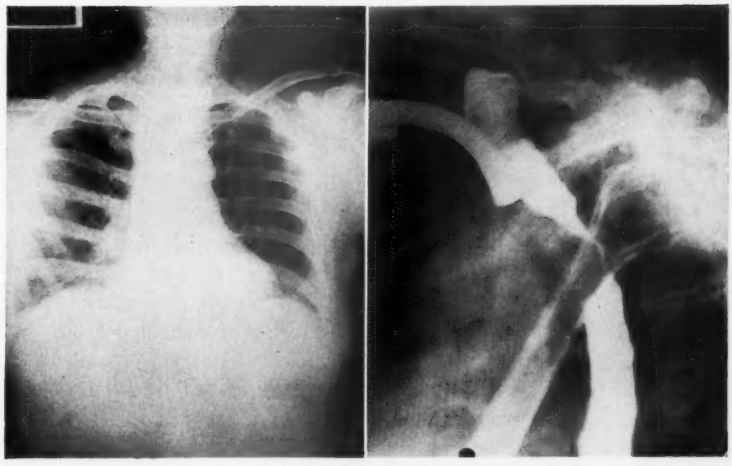

Fig. 8, Case 2.—June 17, 1947—Posterior-anterior view of the chest showing the tracheotomy tube in place and marked widening of the upper mediastinum.

Fig. 9, Case 2.—July 9, 1947—Lateral view of the chest showing distortion of the esophagus with marked constriction at the level of the lesion in the trachea.

esophagus was distorted and greatly constricted at the site of the lesion in the trachea (Fig. 9). This observation suggested that the lesion might be primary in the esophagus.

Histologic examination of the tumor removed from the trachea showed that it was characterized by large, irregular, compact masses of neoplastic squamous epithelium showing no keratinization. These cells were interspersed with a small amount of fibrous connective tissue stroma. The pathologic diagnosis was partially differentiated squamous-cell carcinoma.

Several bronchoscopic procedures were carried out, with removal of tissue and dilatation to provide a freer airway. Roentgen therapy also was instituted. This was administered through a single anterior field, and two anterior oblique fields, 10 cm. in diameter. High-voltage radiation (200 kv), with a filter of 0.5 mm. of copper, at 70 cm. distance was used. The dosage received by the central thoracic field was 2500 r (in air) in 13 days and the oblique fields each received approximately 1000 r (in air) during the following 29

days. Owing to the patient's poor condition and her poor tolerance of radiation, the dosage was inconsistent in both periodicity and quantity and was considered to be therapeutically ineffective.

Since this patient's condition was so critical at the time of admission and the disease so far advanced, it was to be expected that all treatment would be fruitless. Her course was steadily downward and she died on September 24, three months after she was first examined.

Autopsy showed a carcinoma of the trachea with direct invasion of the esophagus. The mucosa of both trachea and bronchi was thick, injected and covered with a foul, purulent exudate. The tracheal tumor was on the posterior surface and extended from just above the bifurcation upward for approximately 3-4 cm. The tumor was firmer than the surrounding tissue and showed ulceration. The portion of the trachea invaded by the tumor was adherent to the esophagus and similar ulcerated lesions were present on the mucosal surface of the esophagus at this same level and 2.5 cm. below. Histologic study of the tumor tissue from both the trachea and the esophagus showed the same structure as that found in the biopsy specimen, that is, squamous-cell carcinoma without keratinization.

Comment.—This case has little interest from the clinical point of view since there was no hope of doing anything that would benefit the patient. It should be noted that at the time this patient was first seen by the otolaryngologist, she was in extreme danger of immediate respiratory failure. In this respect this case of tracheal tumor is similar to others reported by Boëmke and Moritz,[4] Guttman,[5] Cann,[9] Fisher,[12] Dean,[13] Harill,[15] Robin and Mann[16] and Calvet and Girou.[17] Instances in which carcinoma of the trachea had reached a hopeless stage before the patient was examined were described by Brown, Weiss, Iglauer and Fine,[1] Fisher,[12] Dean,[13] Miller,[18] Keeney,[19] Guerra Rousseau, Daumy Amat and Aguirre Medrano[20] and Muta and Ogata.[21] That so many of the relatively few patients with tracheal tumor did not receive skilled medical attention until the patient had symptoms of respiratory collapse or until the malignant lesion was far advanced, or both, certainly indicates the need for persistent efforts to educate both the public and the medical profession regarding the significance of respiratory symptoms that should lead to the suspicion of a new growth in the respiratory tract.

From the pathologic standpoint, this case has a certain interest in illustrating that it may not be possible to determine definitely,

when the lesion is far advanced, the site of the primary growth. Although the pathologist believed that this tumor originated in the trachea, with invasion of the esophagus, the lateral roentgenogram (Fig. 9) certainly was suggestive of a primary esophageal lesion.

DISCUSSION

In preparing this paper it was planned to make a thorough study of the literature that has appeared since Culp's[22] excellent review, published in 1938, of cases reported through June 1936, and to present a definite, up-to-date sum of the cases of primary carcinoma of the trachea. In attempting to do this, numerous difficulties were encountered that make such mathematical exactitude apparently impossible. The chief of these concerns the confusion of terms used by pathologists in describing tumors of the trachea and the disagreement among them as to whether certain lesions are benign or malignant. Until the pathologists agree more thoroughly on the nomenclature and criteria of malignancy of tracheal tumors, it would appear impossible for a clinician reviewing the reports to classify or count them exactly. Another difficulty in trying to determine the number of cases is that sometimes the same case is reported by two different authors, and there is even some discrepancy as to classification and enumeration of cases to be noted in successive reports by different authors from the same institution on the same clinical material. Furthermore, a few articles listed in the Index Medicus could not be obtained for review (Goto and Kyo,[23] Hinojar[24] and Khayutin[25]). The Index Medicus for 1947 is not yet available, so there probably are articles that have appeared during the last year that could not be traced. It is possible also that some articles reporting supposedly benign tumors would contain cases that according to some classifications should be listed as malignant.

For these reasons, the number of cases to be added to the list since Culp's review can be only approximate. Of growths diagnosed as squamous-cell carcinoma, adenocarcinoma and carcinoma of high-grade malignancy, there have been approximately 35 to 40 instances.[1, 2, 8, 12-14, 16, 18-21] In addition, there is a group of about 25 cases, with other pathologic designations, that some might classify as true cancer and others not.[2-6, 8-11, 13, 15, 17, 26-28] Since Culp listed 147 cases of primary carcinoma of the trachea found in the literature to June 1936, the number of cases of this type so far reported is in the neighborhood of 200. It should be remembered also that of the 147 cases listed by Culp as carcinoma, only 82 had histologic verification. In practically all of the cases reported in the last decade there has been a histologic study by competent pathologists, but

even so, numerous questions and confusions remain concerning their exact classification.

The type of lesion about which the most confusion exists is that variously designated as cylindroma, basal-cell carcinoma, mixed tumor of salivary type, cylindric epithelioma and adenoma. If these tumors are not of the same type, the reports about them certainly indicate striking similarities and it would be hard to recognize any criteria for making definite distinctions among them. Some tumors reported as low-grade adenocarcinoma apparently belong in the same general group. Some pathologists even report what appears to be the same type of lesion as sarcoma.

Eggston and Wolff,[29] in their monumental work on Histopathology of the Ear, Nose and Throat, list cylindroma as a benign tumor of the trachea and in discussing cylindroma generally have this to say:

> "A type of composite tumor of the seromucinous and salivary glands has been designated as cylindroma. This tumor is composed of anastomosing cords of broad masses of epithelial cells, surrounding irregular areas or gland-like structures which are filled with epithelial mucus. This should not be confused with the connective-tissue myxoid degeneration which occurs in the stroma. In this type of tumor the cells are suggestive of basal cells of the type of adenoid cystic epithelioma . . .
>
> "Cylindromas may occur in the lacrimal, seromucinous or salivary glands. The present authors see no justification for the continuation of this terminology, as it represents a type of tumor where there are masses of mucoid secretion surrounded by epithelial cells, a common finding in the mixed or composite tumors in the upper respiratory tract. . . . It has been well agreed among students of the mixed tumors of the upper respiratory region that basilomas and cylindromas represent, biologically and clinically as well as from a microscopic standpoint, one and the same tumors and, therefore, it would be well if these terms were dropped."

Eggston and Wolff suggest that it would simplify the situation if all these tumors were spoken of as adenomas of their respective glandular structures.

On the other hand, Tinney, Moersch and McDonald,[2] in reporting a series of 27 cases of carcinoma of the trachea observed at the Mayo clinic, classify cylindroma among the distinct groups of malignant tumor, although their clinical records show that in this group the survival rates far exceeded those for other types of cancer. The Mayo Clinic authors say that cylindroma is sometimes confused with adenoma but presents a different pathologic picture. It tends to produce mucus while adenoma does not. Use of mucicarmine stain may make the distinction. Adenoma tends to be more vascular. Mixed tumor also is sometimes confused with cylindroma

but this has, in addition to epithelial elements, connective tissue which is undergoing myxomatous change.

Another detailed discussion of cylindroma has been presented by Boemke and Moritz.[4] They noted that one school of pathologists has regarded these tumors as of mesenchymal origin, while another has claimed that they are epithelial growths, and still another that they are mixed tumors consisting of both mesenchymal and epithelial elements. These authors offered evidence to prove that cylindromas are of epithelial origin. They emphasized that the clinical literature offers no real information concerning cylindroma of the respiratory organs, since, because of mistaken and confused histologic interpretations, many tumors of this type have been considered as carcinomas or endotheliomas and described as such. They concluded that although these tumors almost always are benign, they often show infiltration and eventually metastases may supervene.

This last statement, which the current reports seem to corroborate, furnishes the clue to the confusion regarding these growths. It would appear that this group of neoplasms, although essentially benign, may undergo malignant change eventually and hence must be considered as potentially, if not actually, malignant. Nevertheless, some distinction should be made between this group of questionable or low-grade malignancy and the more rapidly developing, poorly differentiated types of neoplasm. This distinction is essential, from the clinical standpoint, in evaluating the probable results of treatment.

Although the results of treatment will be least satisfactory in patients with tumors of high-grade malignancy, it should be emphasized that most patients with tracheal tumors of all kinds die as the result of respiratory failure because of obstruction. Any tumor which grows large enough to obstruct the tracheal airway will have a lethal effect on the patient, no matter how benign it may be histologically. Since this is so and since some of the benign tumors apparently are potentially malignant, this again emphasizes the need for bronchoscopic examination of all patients whose symptoms suggest a tracheal obstruction, for prompt and radical removal of any tracheal tumor. If this were generally done, at least some cases of cancer of the trachea should be prevented and the results of treatment, even of tumors that are frankly malignant, should be improved. Since so many of the reported cases of tracheal carcinoma have been so far advanced when the patients were first observed, it is extremely difficult to evaluate the efficacy of treatment in this condition. Results in a few cases, however, including the first case

presented here, would seem to indicate that the chances for prolonging the survival period beyond that usually reported appear at least promising. This hope is strengthened by the fact, which has been noted by all who have written on the subject, that in the majority of cases of carcinoma of the trachea, the metastases are only regional. The only exceptions noted in the recent reports reviewed were the case reported by Keeney[19] in which the patient had widespread generalized metastases even involving the skin and the one described by Fisher[12] in which the patient had metastasis to the bones of the arm. In some of the cases in which the tumors were of high-grade malignancy, death occurred from respiratory obstruction before any metastasis or extension outside the trachea had occurred.

The general facts and features in regard to tracheal carcinoma have been thoroughly discussed by other authors. Hence the details regarding the extreme rarity of tracheal carcinoma in relation to bronchial and other cancers, the dependence of symptoms on mechanical factors incident to the situation of the tumor, age and sex incidence, the predilection of tracheal cancers for the posterolateral walls of the lower third, near the bifurcation and the history of early reports will not be reviewed here.

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XL

TESTING SPEECH-HEARING AND THE EFFICIENCY OF HEARING AIDS

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One should not have to explain the importance of speech-hearing and the testing of it, and yet it is strange to say that after over 25 years of attention by otologists to frequency audiometry, it just now appears that there is general interest in direct speech-hearing testing. This is particularly odd when one is conscious that there always was and always will be an uncertainty in predicting what the speech-hearing loss is in the deaf, when studying their frequency audiograms.

In the early days, Fletcher advised the averaging of the middle range reportings and multiplying this by a factor 0.8. The result was described as "a percentage hearing loss to speech," and was noted on the bottom of the graph. Maico and Sonatone audiometric charts followed suit, and gave curves to indicate percentage loss. However, the findings by these formulae frequently disagreed with the obviously better or worse hearing of the individual tested. Bunch[1] went to considerable length in his book "Clinical Audiometry" to show the futility of attempting to express speech-hearing loss in terms of percentage, and added, "Another method of describing the amount of hearing loss is to state that the audiogram shows an average loss of a certain number of decibels. This method gives as little information as does a statement of percentage loss." These methods of *computing* speech-hearing loss rather than *testing* speech-hearing loss are still perpetuating themselves in the present plan of the A.M.A. Council on Physical Medicine in rating hearing disability. Here, the averaging is guardedly spoken of as the "capacity to hear speech." But still direct testing with speech frequently does not agree with the deductions from the audiograms.

The matter boils down to the fact that the best way to test speech-hearing is by the use of speech as a testing stimulus. This has been practical for a number of years, in fact since 1904 when

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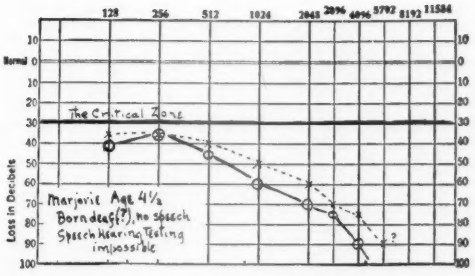

Fig. 1.—Speech-hearing testing impossible. The child was born deaf and the loss was great enough to preclude the normal acquisition of language. There is no auditory word imagery. This audiogram was taken after training in a conditioned response. Later, with auricular word training, a speech-hearing test can be made.

Sohier Bryant first adapted the Edison phonograph to this purpose. There have been a few workers in the testing field who have been enthusiastic on the subject, and each has gotten out his own testing word lists. All have used phonographic speech in order to avoid variable intensities present in the original spoken voice test.*

The instrument needed for modern speech-hearing testing is a phonograph with a motor driven turntable and a crystal pick-up. There are within the box amplifying tubes, and on the front panel is an intensity or loudness attenuator marked in decibels, with a zero setting regulated by a "trimmer." The speech is sent out either over a headphone or over a loudspeaker. The former is used for the individual testing of speech-hearing, the latter for the testing of the efficiency of hearing aids. The record, which is a most important item in the set-up, is one on which numbers are recorded on one side and monosyllabic words on the other. Much care has been taken in the manufacture of these records, first as to the selection of test words and numbers, second as to recording at an even intensity level. It was found advisable to select monosyllables: numbers "one" to "eight" omitting "seven", and monosyllabic words from Thorndyke's list of the most commonly used words in the language. Thus is eliminated the factor of education and intelligence, for the words are in the vocabulary of the most illiterate.

*A bibliography of speech-hearing tests and methods has been compiled by the author.[6, 7]

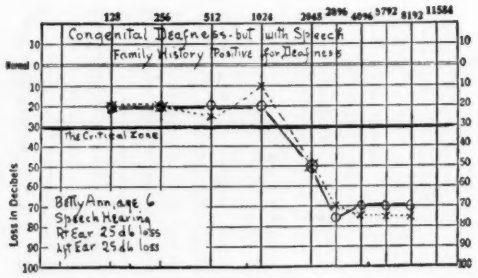

Fig. 2.—Audiogram of a child born deaf but with enough residual hearing to have heard and learned language; she can be tested with the phonograph-audiometer.

In testing the individual ears, the patient is merely directed to repeat the word he hears. The examiner holds a key card to which he refers as to the correctness of the answers. The loudness of the speech is started at a point easily audible and is attenuated to the threshold of hearing. Differing from frequency audiometry where one is often uncertain as to accuracy in the patient's reporting, here the patient either repeats the word or number rightly or wrongly. There is then no doubt about the accuracy.

With phonographic speech from a flat intensity record, there is introduced into the testing a noise comparable to "white-sound" —a flood of simultaneous frequencies in the speech zone.

In all the many fields which deal with audio frequencies and their measurement, one must know definitely what base reference level is used. In speaking of various levels of loudness, upon which much of hearing depends, one must also know whether the loudness is expressed in terms of power, ratios of powers or of energies (decibels), or phons (a newcomer in the field of acoustic terminology). Otologists are nearly always in a state of confusion when explanations of these matters are undertaken. It will be found gratifying and useful to read the clear exposition of terms given by Davis.[2] In audiometric work the decibel has been accepted as the unit of measurement and is "defined as a ratio of two energies or powers. A decibel has no fixed absolute value or any units. It is simply a ratio, telling by what proportion one value is greater or less than the other. To give the decibel scale an anchor, so to speak, we conventionally assume certain reference levels that are understood unless otherwise specified. For acoustic pressures the standard

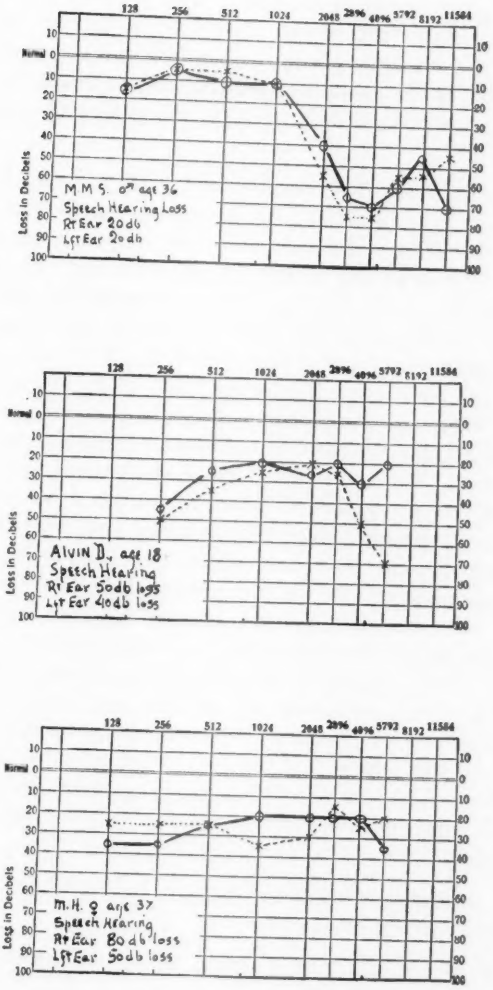
128 256 512 1024 2048 2896 4096 5792 8192 11584
Loss in Decibels
M M S. ♂ age 36
Speech Hearing Loss
Rt Ear 20 db
Lft Ear 20 db
128 256 512 1024 2048 2896 4096 5792 8192 11584
Loss in Decibels
ALVIN D. age 18
Speech Hearing
Rt Ear 50 db loss
Lft Ear 40 db loss
128 256 512 1024 2048 2896 4096 5792 8192 11584
Loss in Decibels
M. H. ♀ age 37
Speech Hearing
Rt Ear 80 db loss
Lft Ear 50 db loss

Fig. 3.—Audiograms of patients whose speech-hearing was worse than the audiogram would indicate.

reference level is 0.0002 dyne per square centimeter. [A dyne is a reference of pressure per surface area, and pressure is proportional to the square root of the power, a watt being the unit of power.] This value of 0.0002 dyne per square centimeter is conveniently close to the intensity of the faintest sound that can be heard by the average ear. Sound that is painful at 140 db. exerts a pressure 10,000,000 times as great."[2]

It seems both practical and logical to adopt for speech work a reference point to correlate with audiometer decibel level. One thousand cycles is the commonest reference frequency used in acoustic work, and it is in the middle of the zone both of speech frequencies and of greatest hearing acuity. The decibel unit is naturally the unit of choice (at this time) to express differences in loudness. The method of setting the decibel meter on the phonograph audiometer has been to use a standard 1000-cycle note record, playing this through the equipment and sending it out over a phone identical (matched) to the phone of the frequency audiometer used for comparison. The 1000-cycle note of the phonograph is matched to the 1000 frequency note of the audiometer at various loudness (db.) levels. One who is wedded to so-called absolute acoustic measurements may object to this method, yet more often than not absolute measurements are to a considerable extent imaginatively "absolute." Further, it may be said that loudness matching, like frequency matching, is an exceedingly accurate phenomenon in the normal ear. "The region of greatest ability to detect small differences in intensity, corresponds to the frequency range of greatest absolute sensitivity of the ear. At 1300 cycles the ear can distinguish the greatest number of tones as being of different intensity, that number being 370."[3] The astounding accuracy of intensity matching is obvious to anyone familiar with the Seashore Musical Talent Tests.[4]

Long experience with speech-hearing testing leads one to accept some common experiences:

1. Numbers are reported at thresholds 10 db. lower than other monosyllabic words.

2. Thresholds are as sharp as with frequency audiometry.

3. The personal equation, or the observational error, rarely amounts to more that 5 db.

4. As with frequency testing, the patient can reach lower thresholds in going from audible to inaudible than in going from inaudible to audible.

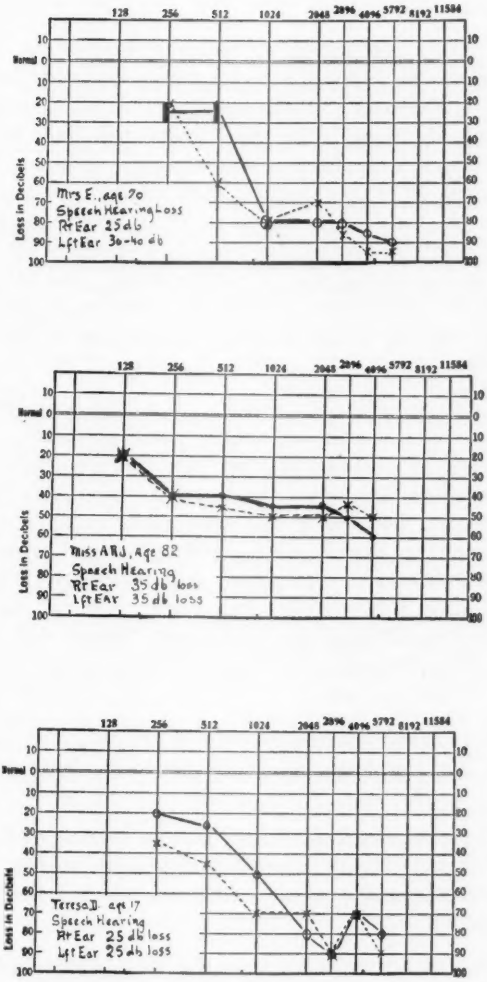
128 256 512 1024 2048 2896 4096 5792 8192 11584
Normal
Loss in Decibels
Mrs E., age 70
Speech Hearing Loss
Rt Ear 25 db
Lft Ear 30-40 db
Miss A R J, Age 82
Speech Hearing
Rt Ear 35 db loss
Lft Ear 35 db loss
Teresa D age 17
Speech Hearing
Rt Ear 25 db loss
Lft Ear 25 db loss

Fig. 4.—Audiograms of patients whose speech-hearing was better than the audiogram would indicate.

5. Very sharp thresholds are seen in those with nerve deafness, less sharp thresholds in those with catarrhal deafness.

6. No other test shows up auditory inattention better than the speech test. In individuals with senile deafness, and in those with profound deafness (50 db. and over) of long standing, inattention usually develops. The phonograph will have to be turned up to a considerable loudness before reporting begins. Then, with the patient exposed to this loudness for a dozen or so test words, the intensity can be dropped as much as 15 db. with the patient still reporting correctly. One might call this a necessity to "warm up" the hearing before testing the "attention deaf." An interesting implication can be made from such findings—the otologist is sure that by attention training he can improve the hearing to the extent that the test shows.

7. In the reporting of words, other than numbers, one will find that the vowel sounds are usually heard correctly. It is in the proper hearing of consonants that mistakes are made. Sentences are not used as testing stimuli except for very particular purposes, since intelligence, familiarity and associations enter in as large factors. Meaningless sounds have been tried and abandoned, because of the difficulty in recognition and reporting. With numbers, some interesting misinterpretations occur. "One" is commonly mistaken for "four" and vice versa; and "three" for "eight", and vice versa. Yet, frequency analysis of these sounds shows no similarities.

8. One should test and record not only the threshold, but the convenient loudness level, which is normally about 40 db. above threshold.

9. The pain threshold should also be investigated. It may be very close to the auditory threshold, in which event it will be difficult to get the patient to accept a hearing aid that gives the needed amplification.

10. Interpretation testing can be done particularly well with this phonograph and word list, and a record can be made of the correct number of answers at the various intensity levels. Silverman[5] has shown definitely that "the threshold audiogram alone is not an adequate measurement of the impaired hearing." And Fowler, Sr. has for some years insisted that our frequency testings of sounds-just-audible left much undetermined. In nerve deafness the efficiency rating does not improve above a certain loudness level, whereas with catarrhal deafness, efficiency increases with loudness

up to the pain threshold. The findings in mixed deafness lie between these two types. Studies should be made to correlate these findings with loudness balance.

11. In testing the relative efficiency of aids, the loudspeaker is used and the patient is placed at a fixed distance from the instrument. He is allowed to adjust the aid to get the greatest efficiency out of it, while the otologist gradually turns down the intensity to the patient's best threshold. The hearing aid modulation controls are tried as well as the loudness control. When an optimum modulation setting is found, interpretation percentage ratings can be made with various loudness levels of the phonograph audiometer. With these two types of testings, the relative merits of hearing aids can be determined.

To start the program of finding the best aid, the patient is merely given the addresses of retailers selling accepted aids; he is told to try the various aids and to bring in those which seem to him to be most satisfactory. The otologist has no concern with keeping on hand an assortment of aids nor with keeping them in good working condition. He has the assurance that the retailer is going to try to give the patient the most suitable instrument. The otologist merely acts as a disinterested third party, selecting by reliable test the best hearing aid for the patient.

In conclusion, it is felt that an enlightened deaf public is expecting better hearing testing work from the otologists, and speech-hearing determination is part of this work. If we do not undertake the obligation, we are to experience the advent of the half-trained lay technician or "audiologist" who will assume a role largely beyond his capacity. At least an otologist should know about degrees and types of deafness; he needs accurate testing for follow-up of treatment, for legally rating disability, for the selection of a proper aid, and for professional guidance and advice. The threshold audiogram alone is not an adequate measure of impaired hearing. Speech-hearing testing cannot be left out of the picture.

1805 CHESTNUT STREET.

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XLI

LERMOYEZ'S SYNDROME—AN ALLERGIC DISEASE

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When an individual has an attack of nausea and vomiting, associated with vertigo, usually of sudden onset, followed by tinnitus and deafness, which usually are progressive, Ménière's disease is the probable diagnosis.

When an individual has an attack of tinnitus and deafness, followed by vertigo, associated with nausea and vomiting, *then* has immediate improvement of the tinnitus and deafness, we probably shall make a diagnosis of Lermoyez's syndrome. These ear symptoms may be unilateral or bilateral in both syndromes but are more often unilateral.

A brief review of the chronological history of Ménière's syndrome is in order to present appropriately Lermoyez's syndrome for analysis. In both diseases we encounter the same symptoms but their appearance is in reverse order to each other, and the prognosis is entirely different. Ménière's disease may cause more or less permanent disability, while Lermoyez's syndrome produces only a temporary disability.

Ménière, in 1861, reported the now famous case of the young lady with an acute infection whom he had treated over a short period of time and who had complained of nausea, vomiting and vertigo, with severe deafness and tinnitus. He, it is now thought, erroneously reported the pathological picture in that case in that he described a hemorrhage into the labyrinth. For many years this one report was accepted as providing the true causation of Ménière's syndrome. However, investigators, not agreeing with this etiology, began to search for other causes. Eventually numerous theories appeared and only recently has there been a unified opinion. Anatomical and pathological material has never been plentiful, and the interpretations on the studies made have been quite varied. The fact that the

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symptoms sometimes appear in young people caused some observers to believe that blood dyscrasias, such as anemias and leukemias, were responsible. This theory presumed that only middle-aged or elderly individuals would provide the arteriosclerotic and atheromatous conditions which could conceivably cause hemorrhages into the labyrinths.

During a later period theories of the cause of Ménière's disease presented the specific or general infections and, then, in more recent decades the vascular anomaly theory appeared. The very recent literature abounds with articles describing hydrops of the endolymphatic system as being the cause of Ménière's disease, and there has been much research work by numerous workers, including Dederding, Lindsay, Fowler, Hallpike and Cairns, and may others, to substantiate this opinion, which is now the accepted one.

During the interim from the older theories to the two latter theories (those vascular anomalies and hydrops of the endolymph sac) there appeared the theory of allergic causation of Ménière's disease and the water and sodium electrolytic metabolism studies by Furstenberg. In 1923 Duke[1] reported in full two cases showing the allergic etiology of Ménière's disease and mentioned three other cases which presented typical symptoms of the disease. These symptoms in his cases could be produced in typical attacks by the ingestion of foods to which the individual was sensitive and, then, the attacks could be controlled by adrenalin. Allergy vaccine therapy was found to produce attacks. Unfortunately, Duke, an allergist and not an otologist, had very little to say about the hearing impairment in these cases, merely stating that in one case the patient had 1/5 depression of hearing. There was no statement as to whether the impairment of hearing progressed or regressed. Duke's report of the allergic basis of Ménière's disease was followed in a few years by numerous other reports, those of Rowe and Richet in 1930, of Proetz,[2] of Vaughan in 1931, of Levy and Vogel in 1932, and in 1933 of Yandell[3] and also Malone.

Proetz, in 1931, reported on an individual who was extremely sensitive to dairy products, especially butter, and who had a very positive reaction to these substances when they were eaten but was not reactive to skin tests. This patient demonstrated a visible patch of edema—allergic in type—in the nasopharynx. This patient had symptoms of Ménière's disease for five years previous to the examination, and he was followed for a period of five additional years. Deafness persisted in this case, indicating a true Ménière's disease. The symptoms were intermittent, a phase of the disease which was

also reported by Crowe in later years. This intermittency seems to depend on exposure to, or the use, of allergens, both in Ménière's disease and in Lermoyez's syndrome.

Yandell and Malone reported orris root as the primary allergen in cases of Ménière's disease. Various other authors found various allergens to be responsible for symptoms. Excellent reviews of the affects of allergy on the inner ear have been presented by Dean[4] and by Hansel.[5] It is quite noticeable and probably of considerable significance that bacterial allergy is scarcely mentioned by any of the writers. Foods seem to have played the predominant role in the production of the symptoms.

In 1941 Atkinson[6] stated that Ménière's disease may be due to more than one cause; in other words, the origin is not always from the same basic condition. From his observations he was able to state that allergy and angiospasm were the two factors which could cause Ménière's disease. He thought that histamine as an allergen was an instigator of many allergic cases of Ménière's disease. He also stated that the histamine test would always be positive in such cases. On the other hand, he found that histamine tests were uniformly negative in the vasospasm cases. In angiospasm cases the patients could be relieved of their symptoms by the use of amylnitrite pearls and acetylcholine, while adrenalin was utilized in producing symptoms, which then could be relieved by nicotinic acid. Atkinson found tinnitus and deafness more pronounced in the vasospasm cases than in the allergic cases, and these symptoms always became worse after the attack of vertigo. In the allergic group, however, he found the opposite—that the hearing impairment and tinnitus improved following the attack of vertigo. His findings, therefore, in the allergic group, coincide with Lermoyez's syndrome in that the appearance of the vertigo was the signal for the improvement of the deafness and tinnitus. Atkinson pointed out that vasospasm occurs sometimes in young people, producing Ménière's disease, and also that in young people the disease may have an allergic origin.

The history of Lermoyez's syndrome is rather brief. In 1927 the late Marcel Lermoyez[7] proposed again the idea of vasospasm, which he had fostered for many years, as the cause of deafness and tinnitus which were followed by vertigo, nausea and vomiting, sometimes by sweating, with all of these symptoms subsiding after the vertiginous attack. He also noted that these attacks may be recurrent. Lermoyez was influenced by the observations of Kobrak as he followed a case of Raynaud's disease, which presented the vasospasm and vasodilation in sequence, and he attempted to correlate

what happened in Raynaud's disease to what may happen in the internal auditory artery. Kobrak had noted these spasm sequences but thought them due to angioneurotic seizures.

Kerekes[8] reviewed all the literature from Ménière's first report up to his own report in 1934. He reviewed the theory of the hemorrhage into the labyrinth, which Ménière had proposed and which was substantiated some years later by Schwabach, in cases showing blood dyscrasias (the anemias and leukemias). Alexander dissented, however, and reported that he found hemorrhage into the labyrinth in no single instance in any of his cases. Manasse had found hemorrhage into the labyrinth in one patient who had vertigo associated with apoplexy. Quincke theorized on local edema producing the pathology in the labyrinth, which theory most closely approximates the hydrops of the labyrinth theory of recent years.

Lermoyez was of the opinion that vasoconstriction involving the internal auditory artery caused the hypo-acuity of hearing and the tinnitus and that when the vertigo appeared it was a sign that circulation had been restored in the artery. With the restoration of the circulation to normal in the artery, all symptoms disappeared and the patient enjoyed normal health again. He pointed out the possibility of spasm in the cochlear artery or vestibular artery individually. Lermoyez is well remembered for his remark relative to the fact that an attack of vertigo due to vasospasm is necessary in the disease he described, for the patient to regain normal hearing. He compares the episode to a violent storm clearing the sky. Throughout the dissertations of Lermoyez and Kerekes there is no mention of allergy as a possible or probable cause of the vasospasm. They were not impressed by any allergic manifestations prior to the symptoms. Kerekes did state, however, that he thought the disease may be due to fundamentally different causes, as Atkinson also stated later, which naturally would permit allergy to be one of these causes. Kerekes reported four cases which he considered to present typical symptoms of Lermoyez's syndrome, all of which he thought to be the result of angiospasm. Portmann[9] in 1929 summarized all the literature relative to vasomotor affections of the internal ear and queried as to whether vasospasm was due to hyperactivity of the sympathetic nervous system or hyperfunction of the adrenal glands.

Let us now return to the remarks of Atkinson. It was stated by him that Ménière's disease could be due to allergy or vasospasm. He found that the hearing impairment and tinnitus were less severe, and then improved or completely disappeared following the attack of vertigo when the disease was due to allergy. These statements

coincide exactly with Lermoyez's recordings, which were affirmed by Kerekes and cited by Nasiell;[10] however, none of these writers named allergy as a basic factor. Is an allergic state the cause of angiospasm? Most authorities on allergy and angiology think not.

We wish to attempt to prove that Lermoyez's syndrome is an allergic disease and that it coincides exactly with the sequence of symptoms and events described by Atkinson, which he claimed produced Ménière's disease, but only when allergic in origin. This is in contrast to the vasospasm origin of symptoms by Lermoyez. Could this reversal in sequence of symptoms as described by Ménière, then, truly be a Ménière's disease? Does not Ménière's disease demand that the deafness and tinnitus follow the vertigo? Would it not seem more logical that Atkinson is describing Lermoyez's syndrome, provided Lermoyez had based his symptoms on an allergic origin? We should like to believe that vasospasm as described by Lermoyez and Kerekes was the result of allergy. We do not wish to classify our cases under the title of Ménière's disease, because the symptoms appeared in reverse order to those in Ménière's disease as we have always known Ménière's disease. Vasospasm of nonallergic origin may easily be an additional or secondary factor following allergy in the promotion of Lermoyez's syndrome, we readily admit, for we have observed some patients who had definite emotional and vasomotor instability. It is to be assumed that the internal auditory artery can be affected quite as well as any other artery in the body, and it is not assuming too much to state that this artery may be involved bilaterally.

Over a period of nine years, our observations in 11 cases, which we believe show symptoms characteristic of Lermoyez's syndrome, have lead us to the opinion that Lermoyez's syndrome is entirely due to, or is associated with, an allergic disease. All 11 patients are essentially well or have been well for a long period of time following their episodes and none has had any permanent symptoms which have incapacitated him, so far as we know.

Our most important reason in claiming allergy as a cause of Lermoyez's syndrome was the consistent finding of urticaria, frequently the giant type, which definitely is an allergic disease. Allergy studies have verified an existing allergic state in some of the patients, while in others who did not have allergy studies we assumed from the history of urticaria given by the patient that the disease was allergic in origin.

We wish to present briefly some of the 11 cases we have encountered demonstrating Lermoyez's syndrome. There have been

instances of recurrence of the disease, and there have been instances of bilateral involvement of the ears. We have been able to follow some of the patients quite satisfactorily, but there have also been some who have not been checked often enough. This is due to the fact that some of the patients resided a long distance from our hospital and only the initial examination could be made. The diagnosis thus depended on the history given by the patient, together with our findings at the time of the examination. Requests to patients to report later developments as to whether they have had recurrence of symptoms or not have not been too satisfactorily complied with.

The 11 patients in our series, which admittedly is a small number, all belong to the white race. The age grouping was of especial interest in that 1 patient was in his twenties, 5 in their thirties, 3 in their forties, and 2 in their fifties. The oldest patient was 55 and the youngest 28. As to sex, 7 were female, and there were 4 males. The ages of the patients in our series of Lermoyez's syndrome is of significance because Ménière's disease is expected to occur more often in the fifties and sixties, and not predominately in the thirties and forties, as we found in our group. The blood serology in all cases was negative.

CASE 1.—V. F. E. aged 39, a physician's wife, was our first patient to have what could be considered Lermoyez's syndrome. The past history was entirely negative for allergy and the patient's personal history had been essentially negative up to the present illness. In September 1938 she had onset of a recurrent swelling, resembling angioneuro-edema, on the right lower leg over the shin. At that time there was much emotional strain and vasomotor instability because of her meticulous caring for a young child less than a year old. After several weeks the condition subsided and then in November 1938 she had pain in the left side and left chest. She had been carrying the baby on her left hip, and because of the size of the baby and her own small stature, she was constantly fatigued. She got along fairly well up to July 1939 when she had giant urticaria involving the entire abdomen and thighs, and in November 1939 there was swelling of both lips, beginning on the upper lip. Then for the first time she noticed tinnitus and impaired hearing in the right ear and she soon began to have dizzy sensations. The most severe dizziness, which approximated vertigo, along with nausea occurred in December 1939. Hearing tests and vestibular tests unfortunately were not done at this time. She then had recurrent swellings of the body involving the right hand and arm, and the right side of the face occasionally felt numb. The dizzy sensations and nausea re-

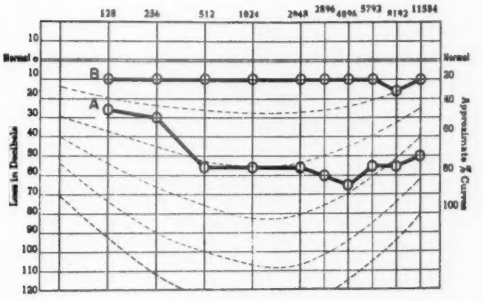

Fig. 1, Case 1.—Audiograms of right ear, *A* made April 2, 1940; *B*, September 19, 1940.

curred. An audiogram was made on April 2, 1940 (Fig. 1), which showed marked loss of hearing of nerve type in the right ear. The hearing had again become much impaired on that date. On that same date she was found sensitive to lipstick and other cosmetics, but all other allergy studies were negative. She apparently had become sensitive to her cosmetics about the time of onset of symptoms the year before, with the recurrent attacks of symptoms as given. By changing her cosmetics, all of the symptoms disappeared and there has been no recurrence to date of any of her symptoms of tinnitus, deafness or vertigo.

This case is an example of sensitivity to orris root or some of the other products in cosmetics, producing urticaria, followed by tinnitus and deafness and then mild vertigo, all of these symptoms appearing in recurrent attacks. With each attack the patient was required to stay in bed, at which time she did not use cosmetics and she would improve or completely recover, only to have a recurrence when she became ambulatory and used cosmetics again. The audiogram made on April 2 was probably taken at a time just previous to the most severe nausea and dizziness. This case further demonstrates that Lermoyez's syndrome may be a recurrent disease with complete recovery between attacks.

CASE 2.—M. G. F., female, aged 38, was first seen on July 12, 1946. This patient stated that three weeks previously, following a ride in a car, she was sitting in a chair at home reading when she noticed a sensation of fullness and tinnitus in the right ear. The ear then felt "dead" and she noticed marked deafness in the right ear. Immediately thereafter she became nauseated and vomited, and

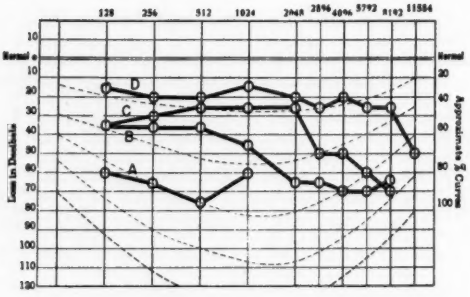

Fig. 2, Case 2.—Audiograms of right ear, *A* made July 12, 1946; *B*, July 15, 1946; *C*, August 16, 1946; *D*, August 25, 1946.

she continued on numerous occasions to vomit over the next few days. She had difficulty in walking. She had a positive history of asthma for the past five years and, in February 1945 she had a severe attack of urticaria. The general history was otherwise negative and she stated that her blood pressure had always been normal. The otolaryngological examination revealed perfectly normal ear drums and quite patent eustachian tubes, with the remainder of the examination entirely normal, except for the right ear in which hearing was almost totally lost. Hearing in the left ear was nomal. The general physical examination, including a neurological examination and laboratory studies, revealed no abnormalities. On subsequent examinations made three days after the first visit, one month later, and six weeks later, the hearing in the right ear was found to show continuous improvement and the tinnitus was decreasing. The graph showing these audiograms for the right ear only demonstrates how much improvement in hearing this patient actually had (Fig. 2). This patient decided to live in Florida and following the last audiogram immediately went to that state and has not been heard from since.

This patient exhibits a solitary but severe attack of Lermoyez's syndrome. She had a history of asthma and urticaria but there was no evident specific allergen causing her attack of Lermoyez's syndrome. Allergy studies were requested but, because of the distance to the hospital and inability to obtain an appointment, were not done. Her recovery of hearing was progressing well during the six-week follow-up period, but even so, her hearing recovery was not so rapid as Lermoyez always described. He expected recovery of normal hearing a few hours after the vertiginous attack.

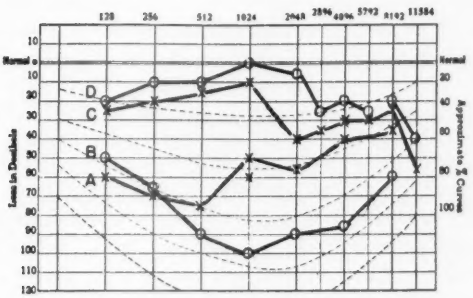

Fig. 3, Case 3.—Curves *A* and *B* made July 26, 1937; *C* and *D*, March 2, 1948. x=left ear; o=right ear.

CASE 3.—W. U. L., male, aged 37, was first seen in 1937. He had a partial, bilateral blindness, since childhood, and in more recent years had been associated with the State Blind Commission in his occupation. When first seen he had marked bilateral deafness and a positive history of being sensitive to various allergens. This patient was seen on numerous occasions, at least annually, following the original visit, and each time he would be aware of recurrent tinnitus and impaired hearing, which in each case were followed by vertigo, nausea and vomiting, with all of the symptoms receding after the vertigo. It was not until some years after he was under observation that we recognized the condition and classified him in the Lermoyez's syndrome group. The patient's history was entirely negative other than for his allergy. This year he consulted an allergist, when the last audiogram was made. The hearing level at the time of the allergy consultation showed a tremendous improvement over the hearing for the original visit in 1937 (Fig. 3), but he states that his hearing is entirely normal between attacks. The first audiogram on the graph probably was made just before an attack of vertigo, and the last audiogram apparently was made just after an attack of vertigo, but before the hearing had completely returned to normal. Presumably his symptoms had been bilateral during all of his attacks. He was found sensitive to a few substances when skin tested and is now only beginning treatment for allergy. We do not know what the future incidence of attacks, if any, will be. The otolaryngological and general examinations of the patient were otherwise entirely negative.

The three preceding cases were taken from our total of 11 cases and they present the various phases of what we consider Lermoyez's syndrome.

SUMMARY

The reversal of the sequence of symptoms in Ménière's syndrome and Lermoyez's syndrome has been presented: namely, in Ménière's syndrome the tinnitus and deafness follow a vertiginous attack and remain stationary or become progressively worse, while in Lermoyez's syndrome the tinnitus and deafness precede the vertigo and diminish or completely subside immediately after the vertigo becomes established. Therefore, there exists a contrasting prognosis for the two syndromes.

Lermoyez attributed his syndrome to a vasospasm of the internal auditory artery without any suggestion of an allergic origin. We have observed 11 patients who demonstrated the typical symptoms of Lermoyez's syndrome, but in each instance we were able to find an allergic disturbance, generally that of urticaria (usually the giant type) sometime prior to the onset of symptoms. We believe the syndrome to be allergic in origin and urticaria probably to be the necessary etiological factor.

Atkinson credited a portion of his cases exhibiting symptoms of Ménière's syndrome to an allergic cause. Those cases coincided precisely in the appearance of the symptoms to Lermoyez's syndrome. We do not think that his cases should have been classified under the title of Ménière's syndrome merely because the group of symptoms was the same as occurs in Ménière's syndrome, since the symptoms appeared in reverse order. It seems no more justifiable to classify his allergy cases under the title of Ménière's disease than it would be to classify a typhoidal or streptococcal infection under the title of diphtheria simply because all of these diseases produce fever, headache and numerous other similar symptoms.

It would seem correct, therefore, to make a diagnosis of Ménière's *syndrome* (as Lermoyez preferred to term it) only when the deafness and tinnitus follow vertigo, and likewise it would seem correct to make a diagnosis of Lermoyez's syndrome in all cases in which deafness and tinnitus are alleviated following the attack of vertigo, with eventual total recovery. We grant that Ménière's syndrome may be allergic in origin, as has been shown by Duke, Proetz and others, or that Ménière's syndrome may be due to vasospasm, as stated by Atkinson. Likewise, we may also state that Lermoyez's

syndrome is due to allergy, as we have attempted to prove, or it may be due to vasospasm as advocated by Lermoyez, Kerekes, and others. But when we then use the terms, Ménière's syndrome and Lermoyez's syndrome, we should be utilizing two terms which really signify separate patterns of symptoms and a definite and different prognosis for each. This would clarify the confusion that exists at the present time and would cause many cases which are now classified under Ménière's syndrome to be rightfully designated Lermoyez's syndrome.

CONCLUSIONS

1. From observations of 11 patients, all of whom had definite allergic manifestations, usually urticaria, and who exhibited symptoms of Lermoyez's syndrome, we believe that Lermoyez's syndrome is definitely an allergic disease.

2. We recognize Lermoyez's syndrome as a separate clinical entity in contrast to Ménière's syndrome and believe that patients who recover normal health after vertigo has cleared their tinnitus and deafness should be classified as having Lermoyez's syndrome.

3. On account of the excellent prognosis of Lermoyez's syndrome, compared to Ménière's syndrome, it is essential that Lermoyez's syndrome be recognized when it exists.

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XLII

THE RESPIRATORY FUNCTION OF THE LARYNX

II. THE ELEMENTARY PHYSIOLOGY OF THE RECURRENT LARYNGEAL NERVE

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AND

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HANOVER, N. H.

In a previous report,[1] we presented graphic recordings of experiments which had to do with laryngeal innervation and vocal cord position in laryngeal palsies. These records substantiated previous opinions of Onodi, Lemere and others, opinions which have been frequently overlooked in clinical interpretations of laryngeal paralysis.

At this time we are reporting work done in an attempt to gain further insight into the physiology of the recurrent laryngeal nerve in order to see why abductor paralysis develops first in recurrent laryngeal nerve lesions or injury; in other words, explain Semon's law.

These experiments, like the previous ones, were done on goats and cats. The recording apparatus in the earlier experiments was identical to that previously reported. In later experiments, two types of transducer were employed to record the pressure changes in the larynx electrically rather than by pneumatic apparatus. The laryngeal tube-balloon apparatus remained unchanged. To the small tube (or tubes) connected to the laryngeal balloon(s), we have attached a Statham strain gauge in some instances, and in others piezoelectric pulse-recording pick-ups. The output of the strain gauge was connected to the string of an old-fashioned string galvanometer electrocardiograph and the movements of the string photographed. The crystal pick-ups were connected to a Grass Model III electroencephalograph and the pressure changes recorded by the ink writers. The form of record using the strain gauge is identical

Read before the Eastern Section Meeting of the American Laryngological, Rhinological and Otological Society, New York City, January 16, 1948.

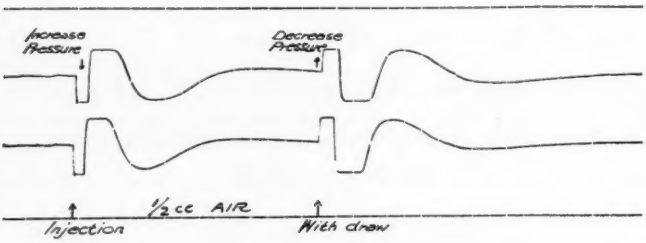


Fig. 1.

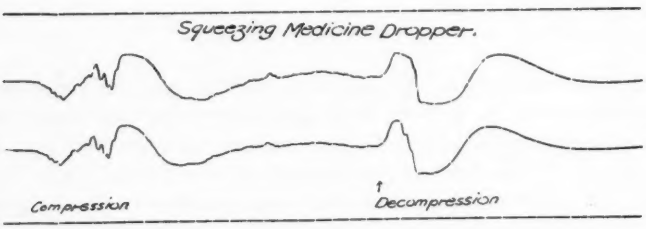


Fig. 2.

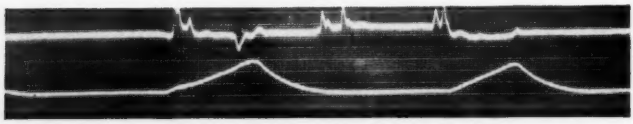
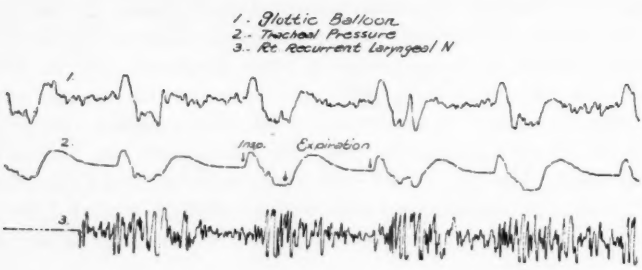


Fig. 3.



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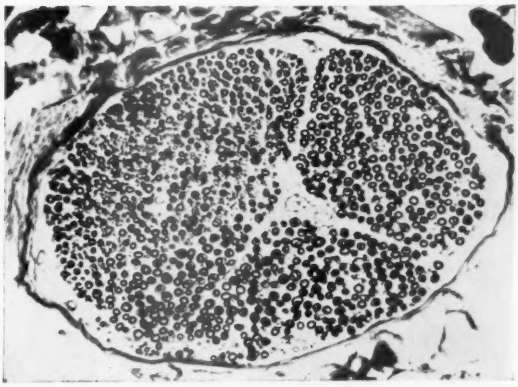


Fig. 5.—Photomicrograph of cross section of human recurrent laryngeal nerve obtained immediately after death and placed in osmic acid. (The specimen was prepared for us by the Department of Pathology, Mary Hitchcock Hospital.)

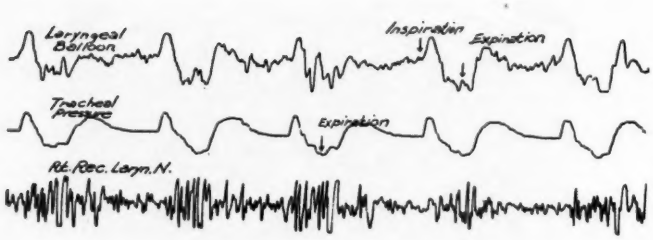
to that previously obtained in the original method. With the crystal pick-up, the pressure records are much modified and can best be understood by seeing some actual records obtained by injecting air into the recording system (Figs. 1 and 2). The records are self-explanatory and should make clear that what is recorded is not pressure but rate of change of pressure; i.e., a steadily maintained pressure records as zero change.

We have attempted to evaluate recurrent nerve sensitivity by obtaining the chronaxie of its fibers and by using alcohol and cold block. The chronaxie was obtained by stimulation of the nerve, using condenser discharge and doing the required calculations or using rectangular electrical stimuli from an electronic apparatus designed for that purpose. When we attempted to secure by electrical stimulation either adduction or abduction of the vocal cords, the same electronic device that was used in chronaxie determinations was employed by changing the apparatus to give repeated stimuli instead of a single stimulus.

Action potentials of the recurrent nerve were recorded on the electro-encephalograph.

RESULTS

At no time were we able to secure abduction of the vocal cords using single shocks applied to the recurrent nerve. Adduction was invariably the only observable or recorded reaction.



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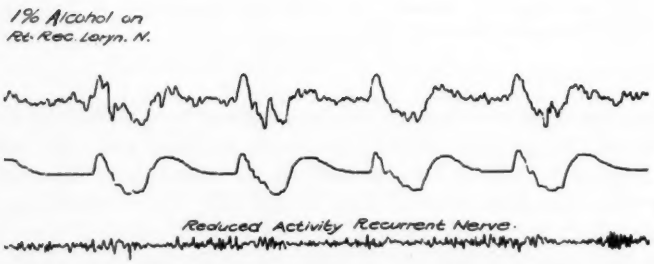

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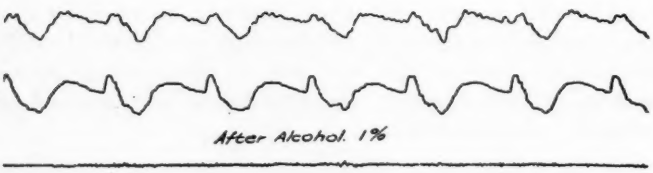

Fig. 8.

TABLE I

Tabulation showing circumstances under which ABDUCTION of the vocal cords occurred in the anaesthetized goat.

FREQUENCY Stimuli per second	VOLTAGE	DURATION Stimuli in m.secs
17	40–80	0.2–0.5
5	1.6	0.01
10	1.6	0.01
20	1.6	0.01
40	10.0	0.01
80	10.0	0.01
14–140	10	20.0 *
40	12	1.0
40	10	1.0
40	4	1.0

(*) Stimulating nerve to post crico arytenoideus muscle.

Using repeated shocks, abduction was obtained infrequently (Table 1).

Abduction has been produced by sliding a suture back and forth under the recurrent nerve (Fig. 3).

Results typical of the general run of experiments may be seen in the protocol of May 7, 1946. The animal used was a 14 kilo goat which was given 21 cc. of 3% pentobarbital sodium in 10% alcohol. Ether was used during the operative procedure which exposed the left recurrent nerve. Repetitive unidirectional stimuli were used with the results shown in the tables. The various factors yielding adduction are shown in Table 2A. It is to be noted that at high intensities respiration was stopped. This has been noted on several occasions. It may be because of a spread of the stimulus to the vagus proper and not actually represent recurrent activity. The occasions when repetitive stimuli gave abduction in this experiment are shown in Table 2B. These results are fairly typical of those obtained in other experiments. As yet, however, we are not able to produce predictable abduction.

Figures 4 and 6-8 show the type of record obtained in studying the action potentials of the recurrent nerve using the Grass electroencephalograph as the recording instrument.

TABLE 2 A

Extract from Protocol of Experiment of May 7, 1946

FREQUENCY Stimuli per second	VOLTAGE	DURATION Stimuli in m secs.	REACTION
8	0-14	5	sl add (s)
6	0-14	5	sl add (s)
5	0-20	5	sl add (s)
100 → 500	1	5	add
100 → 500	10	5	add
17	14	20→001	add
17	20	001 → 20	sl add
17	40	001 → 1	?
100	0→10	5	add
100	10→100	5—50	add
100	10→100	50—100	stopped resp.
80	0—10	5	add
60	0—10	5	add
40	0—14	5	add
20	0—14	5	sl add
15	0—14	5	sl add
10	0—14	5	add (s)

(s) Single twitches

TABLE 2 B

Extract from Protocol of Experiment of May 7, 1946

FREQUENCY Stimuli per second	VOLTAGE	DURATION Stimuli in m secs.	REACTION
17	40	2	opp. abd.
17	40	5	opp. abd.
17	40	10	?
17	40	2	abd.
17	40	5	sl. abd.
17	40	2	sl. abd.
20	40	2	?
17	40	2	?
17	50	2	sl abd.
17	80	2	abd.
17	80	2	? *
10	40	2	?

(*) Nerve may have been permanently injured by heat from very high voltage stimulation

TABLE 3

	NERVE	MUSCLE	NERVE	MUSCLE	MUSCLE
	Superior Laryngeal	Crico Thyroid	Recurrent	Post Crico Arytenoid	Thyro Arytenoid
Cat	$4 \rightarrow 8 \times 10^{-4}$	$3 \rightarrow 9 \times 10^{-4}$	2×10^{-5}	—	—
Goat	—	—	1×10^{-5}	1×10^{-4}	—
Goat	—	—	2×10^{-5}	2×10^{-4}	—
Goat	4.5×10^{-4}	2×10^{-4}	2×10^{-5}	5×10^{-4}	—
Goat	7×10^{-4}	—	—	—	—
Cat	—	1×10^{-4}	2×10^{-5}	1×10^{-4}	2×10^{-5}
Time (Chronaxie) in seconds					

From the results we have so far obtained, it would appear that the greatest activity in the recurrent nerve occurs just before and early in the expiratory phase of respiration. Alcohol block profoundly reduces the recorded activity and shows that the electrical changes observed are probably real. The number of nerve fibers involved in abduction would appear to be few when compared with the number involved in adduction.

At no time during any experiment using nerve block (alcohol or cold) have we been able to suppress adduction and secure abduction. This held whether one was merely observing the motion of the vocal cords or attempting to produce abduction by electrical stimulation. It was expected that the smaller and less heavily medullated fibers would be more sensitive to blocking agents. Lemere[2] considered that in some cases at least the adductor muscle fibers and the nerve fibers supplying them were smaller than those involved in abduction. The evidence which we have obtained seems to be exactly the contrary.

It is generally assumed that small, thinly medullated nerve fibers have a longer chronaxie than larger, more heavily medullated ones. Our chronaxie results are illustrated by Table 3, which summarizes the information gained from a number of experiments.

The evidence in this table would indicate that in the cat and the goat the motor fibers of the superior laryngeal nerve are of about the same size. We know from previous experience that this nerve

supplies only the cricothyroid muscle. The cricothyroid muscle with its nerve supply not degenerated has the same chronaxie as the nerve.

In the recurrent nerve the situation is different. We know that it supplies abductors as well as adductors. The chronaxie of the nerve, as we have obtained it, is about the same in both cat and goat. The chronaxie of the posterior crico-arytenoid (an abductor) with its nerve supply intact is about ten times as long as the time obtained for the recurrent nerve. This time for the nerve agrees with the chronaxie of the thyro-arytenoid muscle (adductor). Apparently there are two general orders of fiber running in the nerve, some relatively large and heavily medullated supplying adductors and some relatively small supplying abductors. These abductor fibers must be few in number and when one is dealing with the intact nerve (stimulation or block), they must be completely submerged by the larger more numerous fibers (Fig. 5).*

CONCLUSIONS

1. The greatest activity in the recurrent laryngeal nerve occurs just before and early in the expiratory phase of respiration.

2. The greater number of fibers in the recurrent laryngeal nerve are relatively large and supply adductor muscles.

3. There are some few fibers supplying the abductor muscle which must be smaller and probably less heavily medullated. Presumably these would be more readily injured by chemical or mechanical agents.

4. We have thus far been unable to obtain by controlled electrical stimulation of the recurrent nerve predictable abduction of the vocal cords.

DARTMOUTH MEDICAL SCHOOL.

*Since this paper was read, there is some evidence that abductor and adductor function is anatomically separate in the recurrent laryngeal nerve of the cat.

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XLIII

EPIDERMOID-LIKE LESIONS OF THE FRONTAL BONE SIMULATING MUCOCELE

REPORT OF TWO CASES

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AND

JOSEPH X. MEDWICK, M.D.

ROCHESTER, MINN.

Mucocele of the frontal and ethmoid sinuses is not a common condition, nor is it a rare one. Given a patient who presents the history of slow, painless displacement of the orbital contents, with or without a preceding history of sinusitis, and who on examination is found to have a soft, fluctuant or doughy tumor attached to the roof or nasal aspect of the orbital wall but not attached to the bulbus oculi, with expansion of the bony external table or floor of the frontal sinus, and roentgenologic evidence of absorption of the bony walls and intercellular septa of the frontal and ethmoid sinuses, the rhinologist readily makes the diagnosis of mucocele of the frontal and ethmoid sinuses.

Benign epidermoid cysts or tumors occurring in the region of and involving the orbit may present strikingly similar clinical and roentgenologic findings and may be confused with mucocele on preoperative diagnosis. Two such examples have been observed in recent months; in each instance the preliminary diagnosis was mucocele of the frontal sinus. Operative findings in each case revealed cheesy masses of desquamated epithelium which grossly resembled the cholesteatomatous material found in chronic mastoiditis. These cases will be reported in detail.

Muller,[1] in 1840 described new formations in the temporal bone composed of concentric layers of epidermoid cells with cholesterin crystals between them. Virchow[2] called this "the pearly tumor." Kaufmann[3] gave its location as the fibrous coverings of the brain, principally the vascular pia, the pericranium and the bones of the

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skull, as well as the testes. Kaufmann,[3] in discussing the etiology of cholesteatoma, quoted both Virchow, who assumed the tumor to be of endothelial origin, and Ziegler, who believed it to be an epithelial structure of epidermoid origin. Kaufmann believed that in the majority of cases there was an early snaring-off of epidermis, in which event these tumors would be dermoids or dermoid cysts.

Spencer and LaRue[4] discussed the origin of cholesteatoma and distinguished true cholesteatoma, which they described as a disturbance of evolution during embryonic life, from false cholesteatoma, which is the encroachment of epithelium from without into a sinus cavity. They reported on a patient from whose orbit and frontal sinus a cholesteatoma, 5 x 7 cm., was removed. They believed this lesion to be a false cholesteatoma, although the manner of entry of the squamous epithelium into the frontal sinus was not made clear. Spencer[5] later reported that this patient died of carcinoma which developed at the site of the cholesteatoma.

Otrich[6] reported a case in which cholesteatoma involved the antrum, the ethmoid bone and the middle turbinated bone. There was no history of trauma. Hayden, discussing Otrich's paper, mentioned a cholesteatoma of the frontal sinus which followed a Killian operation.

Lillie[7] found a large, foul-smelling cheesy mass resembling cholesteatoma in the frontal sinus of a man who had undergone an extensive intranasal operation eight years before.

Geshelin[8] reported a case in which a draining fistula had been present in the right frontal sinus after injury eight months before. At operation the right frontal sinus was found to be filled with granulations. The intersinus septum was absent, and cholesteatoma-like material was found in the left frontal sinus. Geshelin found nine cases of "epidermoids" of the frontal sinus reported in the literature. He was of the opinion that cholesteatoma is the result of implantation of squamous epithelium into the sinus cavity, and quoted Lautenschlager, who believed that it is impossible for sinus mucosa to form cholesteatoma, even in the presence of inflammation.

Frostberg[9] reported one case in which cholesteatoma formed in the frontal sinus, and stated that it was impossible to distinguish a cholesteatoma from a mucocele or a pyocele by roentgenologic examination.

Coates,[10] in 1937, reported a case of orbital abscess secondary to chronic inflammation of the frontal sinus. The patient had suf-

fered a penetrating injury to the orbit 35 years before. At operation, erosion of the outer table and floor of the frontal sinus was found; lateral to the sinus was found an area of erosion in the frontal bone, with a mass of cholesteatoma between the frontal bone and the dura, which showed inflammatory thickening. Coates included notes on the condition of a patient observed by Woodward. Woodward's patient complained of severe, sudden pain over the right eye of 17 days' duration, with swelling of the eye. Frontal headache had been noted for nine months. Roentgenograms showed a large area of decreased density in the right frontal sinus. A mucocele was suspected. At operation, necrotic membrane and free pus were encountered, and lying free in the sinus was a mass of cholesteatoma, 2 x 3 cm.

Lillie and Pastore,[11] in 1941, reported two cases of hemorrhagic cysts, which simulated mucoceles, in the frontal sinus. In each case the presenting complaint was displacement of the eye. Both patients presented a history of pain in the affected sinus. One patient had been kicked in the head by a horse some 20 years before. Roentgenologic examination of each patient revealed an area of rarefaction in the frontal bone lateral to the frontal sinus; at operation, the frontal sinus was found not to be involved. Both cysts contained fluid and amorphous material with cholesterin crystals, and both were considered to be the result of hemorrhage. The walls of the cysts exhibited no characteristic histologic structure in either case. These two cases are similar to the cases to be reported.

REPORT OF CASES

CASE 1.—A woman, 33 years old, registered at the clinic on June 16, 1947. She had noticed fullness and drooping of the right upper eyelid in May 1946. This deformity had been progressive, with downward displacement of the eyeball. In April 1947, a small lump had been noted in the right eyelid, 1 cm. above the outer canthus. Three weeks before the patient's admission a lump had been noted below the right ear. Normal pregnancy had been terminated in May 1947. There was no history suggesting sinusitis at any time in her life and no history of trauma to the face or head.

Examination of the nose showed no abnormality. The right palpebral fissure was narrowed and the right eyeball was displaced downward. A small, flat, freely movable mass was noted in the upper temporal quadrant of the right orbit; a soft, doughy tumor was felt by deep palpation at the same area. A small mass, 1 cm. in diameter, was noted behind the ramus of the right mandible. The ophthal-

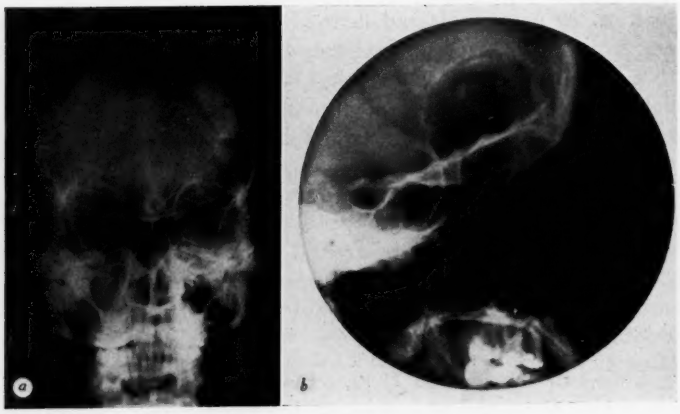

Fig. 1, Case 1.—Roentgenograms of the skull; *a*, anteroposterior view, *b*, lateral view.

mologist reported no abnormalities except slight exophoria with the Maddox rod test. Results of a general physical examination were essentially normal.

Roentgenograms of the paranasal sinuses revealed a large area of decreased density lateral to the right frontal sinus and apparently not continuous with the sinus. There was evidence of involvement of the roof of the orbit and the floor of the anterior cranial fossa on the right (see Fig. 1*a* and *b*). Results of other laboratory examinations were essentially normal.

On June 24, 1947, with the patient under general anesthesia, the mass below the right ear was removed. The pathologist reported this to be adenocarcinoma, mixed tumor type, of the parotid gland. This growth was encapsulated. Incision was then made over the lateral aspect of the right supraorbital ridge, below the brow, and carried down to the bone. When the periosteum was elevated from the roof of the orbit, a dehiscence in the bone was noted. A soft mass covered by a white membrane bulged through this opening into the orbit. On incision of the membrane, flakes of material resembling cholesteatoma were found. The cholesteatoma matrix was then separated, and the overlying bone was removed, so that the entire mass, which measured 4 x 5 cm., was uncovered. The mass was removed within its capsule (Fig. 2*a*). Medial to the mass, the

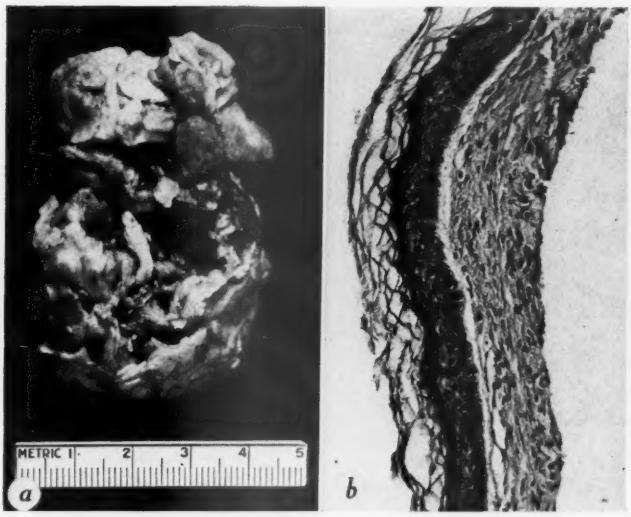

Fig. 2, Case 1.—*a*, Mass removed; *b*, photomicrograph of section showing squamous epithelium which lined the wall of the cyst. (Hematoxylin and eosin stain; x 138)

dura was exposed and displaced posteromedially. A lacy network of bone, which had not been completely absorbed, remained attached to the dura. This bone was removed. The frontal sinus was not entered during the operation. Two soft rubber tubes were placed at the lateral extremity of the incision and the wound was closed.

After removal of this mass, search was made for the small nodule in the eyelid, but nothing was found.

The pathologist reported that material from the frontal region consisted of desquamated epithelial cells and cholesterin. The capsule was reported as consisting of a thin cyst wall lined by keratinizing squamous epithelium (Fig. 2*b*).

The postoperative course of the patient was afebrile. Drains were removed on the fourth postoperative day and the patient was dismissed from the hospital on the sixth postoperative day.

CASE 2.—A 39-year-old man registered at the clinic in August 1946. He stated that he had first noticed swelling of the left upper eyelid three years before. One year later he had noticed diplopia.

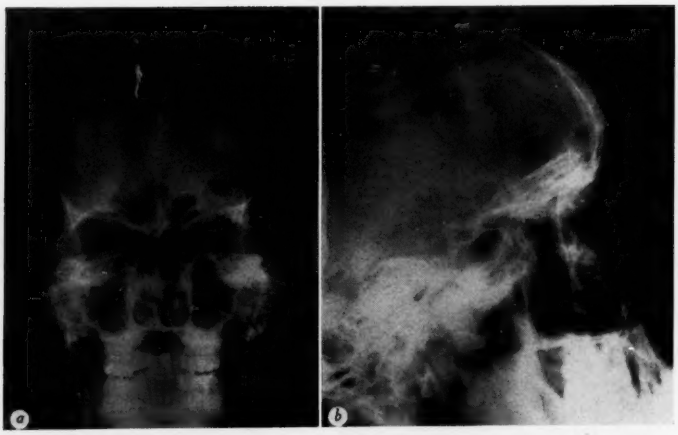

Fig. 3, Case 2.—Roentgenograms of the skull; *a*, anteroposterior view, *b*, lateral view.

The history revealed no symptoms suggesting infection of the paranasal sinuses. The nose had been broken in an accident during the patient's boyhood.

The ophthalmologist reported that the left eyeball was pushed downward and lateralward by a mass arising from the roof of the left orbit. Rotation of the left eyeball was limited in all directions. Examination of the nose revealed a flattening deformity of the nasal bones and fracture of the septum. There was no evidence of infection of the paranasal sinuses. Results of a general physical examination and laboratory studies were noncontributory. Roentgenograms of the paranasal sinuses and orbits revealed an area of decreased density in the left frontal bone, lateral to the left frontal sinus, with involvement of the roof of the orbit (Fig. 3*a* and *b*). The diagnosis was mucocele of the left frontal sinus, and surgical treatment was advised. The patient elected to defer operation at this time. He returned in August 1947 for operation. No change in the appearance of the tumor had occurred during this interval.

On August 9, 1947, with the patient under general anesthesia, an incision was made over the lateral aspect of the left supraorbital ridge, inferior to the eyebrow. When the periosteum was elevated from the roof of the orbit, a brown, cyst-like mass, 2 x 2 cm., was encountered. Above this mass, at the midpoint of the orbit, the

Fig. 4, Case 2.—*a*, Mass removed; *b*, photomicrograph of inflammatory tissue from the wall of the cyst. Note absence of epithelium. (Hematoxylin and eosin stain; x 18)

frontal bone was markedly thinned. This bone was removed, so that a thin sac was exposed; this sac was filled with a mass of cheesy material bathed in a brownish fluid. The separate lobules of the mass were more or less adherent, one to another. The lobules had pitted the bone which formed the walls of the cavity. The orbital cyst was ruptured while it was being removed. Thick, brownish fluid containing cheese-like material was evacuated. The cyst was removed in its entirety (Fig. 4*a*). The pocket in the frontal bone was smoothed and the wound was closed with a Penrose drain. The frontal sinus was not entered. The pathologic report of the tissue removed was: "Cyst lined by inflammatory tissue with blood pigment and small calcareous deposits in its walls. Careful search of several sections did not reveal any epithelial lining in the cyst" (Fig. 4*b*). Material from the cyst was reported as containing blood pigment and cholesterin.

The patient's postoperative course was afebrile. He was dismissed from the hospital on the third postoperative day and from our care one week later.

Comment.—In the two cases presented, the clinical findings were strikingly similar. Both patients exhibited a clinical picture

typical of mucocele of the frontal sinus, with erosion of the roof of the orbit and displacement of the orbital contents. Neither presented a history suggesting chronic sinus infection or nasal allergy. Neither had suffered pain in the affected area. One patient could recall no trauma; the other had suffered fracture of the nasal bones without a penetrating injury many years before the onset of symptoms. Examination of the nose and pharynx revealed nothing significant in either patient. Roentgenograms in each case showed the sinuses to be normal in appearance, with an area of rarefaction in the temporal bone lateral to and separate from the frontal sinus.

The operative and pathologic observations differed in certain respects. In the first case, a single membranous sac was found which was filled with odorless, amorphous material arranged in small masses. This material, when examined microscopically, was shown to be composed of epithelial cells and cholesterin crystals. The thin-walled sac was lined with stratified squamous epithelium. This specimen was similar to the cholesteatoma and matrix that are found in cases of chronic mastoiditis.

The second patient presented both a thick-walled cyst, filled with a brownish, syrupy fluid which contained cheesy material, and a thin-walled sac containing amorphous material grossly similar to that found in the first case. Microscopic examination of this specimen showed blood pigment in both the fluid of the cyst and the cholesteatoma. No epithelium was found lining either sac. Actually, the epithelial origin of this specimen has not been demonstrated, and accurate histopathologic classification of the lesion is difficult.

The etiology of these lesions is obscure. Since each was separated from the frontal sinus by a solid bony wall and no connection with an aberrant ethmoid cell was demonstrated in either, the sinuses can be eliminated as a factor in the development of the lesions. The different histologic structure of the two specimens suggests that they were not caused by the same factors.

The epithelial nature of the lesion in the first case was clearly demonstrated. In the absence of a history of trauma or surgical treatment which might have implanted squamous epithelium into this region, it seems reasonable to postulate that the origin of this lesion was an epithelial cell rest which occurred during embryonal life. The presence of a filigree of thin bone between the sac and dura, and the thin layer of bone separating the sac from the integument suggest that the lesion originated within the frontal bone. A cell rest would appear to have been embedded in the diploic squam-

ous portion of the frontal bone. Kaufmann[3] wrote that true cholesteatomas are found in the bones of the skull. This case represents, in our opinion, an example of true cholesteatoma occurring within the frontal bone.

The specimen in the second case contained blood pigment, but no epithelium. Both parts of the specimen were enclosed by a wall of fibrous tissue, of varying thickness, which could be separated readily from surrounding tissues. The two sacs which composed the specimen varied slightly in the character of the wall and in content. One sac, with thick walls showing evidence of inflammation, contained brown fluid and flakes of cheesy material. The other sac had thin walls and contained primarily cheesy material bathed in brown fluid. The content of both sacs was composed of blood pigment and cholesterin. Either of two sources for this lesion can be postulated, although the findings do not fully confirm either theory. First, it might be said that hemorrhage followed trauma and that the effused blood subsequently became encapsulated. The interval of many years between the time of trauma and the first appearance of symptoms does not favor this theory. Second, it might be argued that the lesion was a primary cholesteatoma in which inflammation had destroyed the epithelial lining of the cavity and caused hemorrhage into the cavity. Although there was no history of inflammation in this region, this theory seems more tenable to us.

MAYO CLINIC.

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XLIV

THE ACOUSTIC PROBE

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The ophthalmologist would be subject to criticism if he made no attempt to use corrective lenses following a cataract operation, even though the vision in the other eye was adequate. Yet this is the usual procedure for the treatment of chronic otitis media after a radical mastoidectomy has been performed, despite the fact that the individual may have excellent bone acuity. The treatment of the infection has been stressed at the expense of the patient's hearing; if a radical mastoidectomy is performed, it is considered a life-saving operation and is not done because of any possible improvement in hearing which may result.

Endaural surgery has improved our knowledge of temporal bone anatomy so that the otologist approaches the radical mastoidectomy with confidence and is now able to obtain a dry, healed cavity in a high percentage of cases. Unfortunately, however, he usually leaves a small stenosed canal that could not accept a prosthesis which would restore the hearing to a serviceable level.

The endaural incisions for a radical mastoidectomy have been carefully described by numerous surgeons stressing adequate exposure without injury to the cartilage. This fear of perichondritis is difficult to understand. Prior to the introduction of the sulfonamides and the antibiotics, perichondritis was a rare complication when all radical mastoidectomies were performed by the postauricular route in which the cartilage was always cut.

A previous report stressed the use of the acoustic probe in chronic otitis media to determine whether a contact area is available for the artificial middle ear.[1]

The acoustic probe is a simple, efficient sound conductor which may amplify the examiner's voice by as much as 50 decibels. It consists of a diaphragm which supports a slender stem of bamboo tipped with a small ball. The diaphragm may be made of plastic, x-ray film or thin cardboard.[2] The probe is patterned after the bird's middle ear, which is quite similar to that of the mammal ex-

cept that the ossicular chain is represented by a simple stick-like structure known as the columella[3] (Fig. 1).

A radical operation should be considered for all patients with chronic otitis media with nonserviceable hearing, providing a proper contact area for the prosthesis has been established.[2] If the patient has adequate bone acuity, the usual incisions should be modified so that a large portion of the concha is removed. The overlying skin of the concha should be preserved to prevent excessive granulations and possible stenosis. It is impossible to attempt to fit the ear canal with a prosthesis if the external orifice is smaller than the radical cavity (Figs. 2 and 3).

The acoustic probe should prove a valuable aid in the diagnosis of otosclerosis. Tuning fork tests to determine whether there is

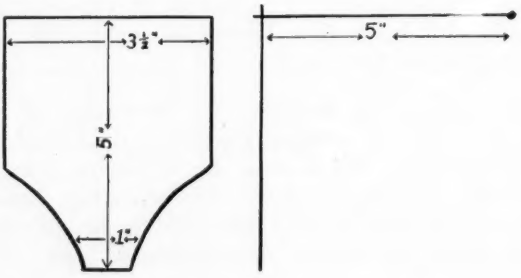

Fig. 1.—Diagram of acoustic probe made with thin cardboard.

fixation of the stapes have never been very satisfactory or extensively used by the otologist. The probe now offers a positive test as to the mobility of the stapes. The short process of the malleus is lightly touched with the probe while the examiner keeps talking. If his voice suddenly becomes loud to the patient, it means that the oval window is functioning and that the conduction deafness is due to other causes. If there is no reaction, the stapes is fixed (Fig. 4).

Patients who have undergone the Lempert fenestration operation with no improvement in hearing or who have obtained an improvement that is below the useful serviceable level should be examined with the acoustic probe to determine whether they are candidates for the diaphragm rod prosthesis.[1] Unfortunately, the majority of the patients who reacted positively to the probe could

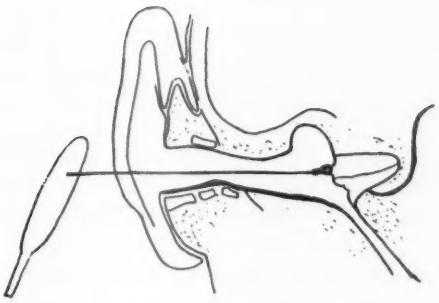

Fig. 2.—Drawing showing the acoustic probe in contact with the stapes in a case of chronic otitis media.

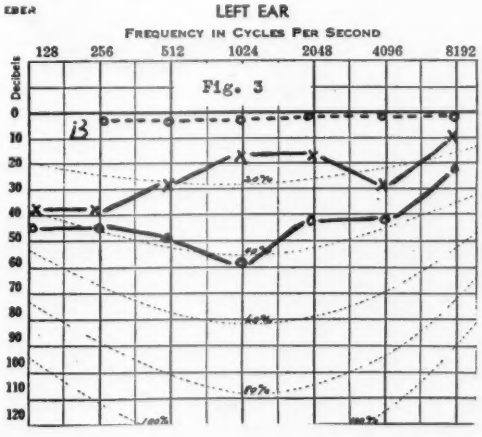

Fig. 3.—Audiogram showing the effect of a middle ear prosthesis in a radical mastoid cavity.

not be fitted with a prosthesis because of a stenosed canal. The possibility of leaving a suitable postoperative cavity should be considered by the surgeon, as a dramatic improvement in hearing has been obtained when the patient could accept a prosthesis to function as an additional transformer over the surgically created yielding area of the fistula (Figs. 5 and 6).

Examination of the fistula with the acoustic probe may yield valuable information as to why patients with excellent bone acuity did not show an improvement in hearing following surgery. The majority of these patients had a positive reaction to the probe, indicating that the fistula was patent and that osteogenesis was probably not the offending factor. However, the flap was found to be

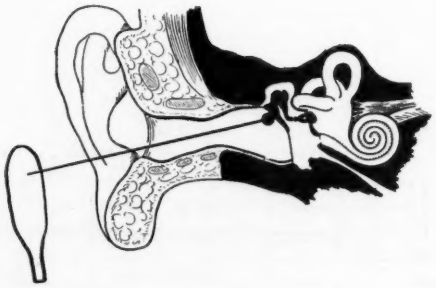

Fig. 4.—Drawing showing the acoustic probe in contact with the short process of the malleus to determine whether the stapes is fixed.

thickened and doughy in consistency and actual tenting was visible when contact was made with the probe, demonstrating that the flap was possibly acting as an insulator rather than a transformer for sound to the fistula. If this is true, then the acoustic probe should help the otologist decide whether a revision of the flap is indicated in unsuccessful fenestration cases.

SUMMARY

Every otologist should be equipped and familiar with the use of the acoustic probe in the management and treatment of deafness.

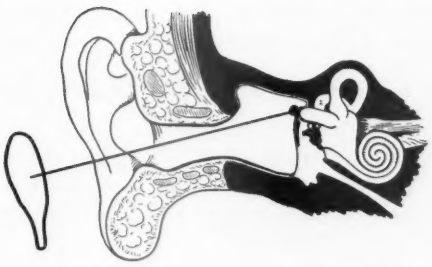

Fig. 5.—Drawing showing the acoustic probe in contact with the artificially created fistula in an ear which has undergone a fenestration operation.

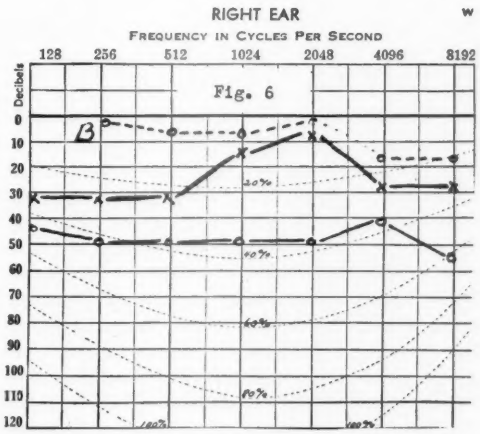

Fig. 6.—Audiogram showing the effect of a middle ear prosthesis on a fenestration case in which there was no appreciable improvement in hearing following surgery.

The acoustic probe should be employed on all patients whose drum membrane and ossicles are absent or have been removed to determine whether a contact area for the artificial middle ear is available.[2]

The acoustic probe will positively determine whether there is fixation of the stapes.

It should prove a valuable diagnostic aid in the management of cases in which the Lempert fenestration operation has given no improvement in hearing or improvement that is below the useful serviceable level.

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XLV

INCIDENCE OF OTITIS MEDIA AND MASTOIDITIS IN SCARLET FEVER

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AND

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The purpose of this paper is to present a statistical analysis of the incidence of scarlet fever and the incidence of otitis media and mastoiditis complicating scarlet fever by years and by age in 18,659 cases of scarlet fever, covering a 23-year period from 1923 to 1945, inclusive. The effect of chemotherapy will also be discussed.

It should be emphasized that the tympanic cavity or middle ear is an extension of the pharyngeal cavity. According to Arey,[1] each auditory tube and tympanic cavity represents a drawn-out first pharyngeal pouch and appears in embryos 3 mm. long. Each pouch undergoes actual constriction and lengthening to form the auditory tube, the outer end enlarging into the tympanic cavity. The pneumatic cells of the mastoid bone are formed by the progressive outgrowth of air cells from this cavity into the solid temporal bone. The ossicles, their muscles and the chorda tympani nerve all become invested with the epithelium of the tympanic cavity and are therefore continuous with the lining of the nasopharynx.

Only a brief review of the anatomy of the middle ear, auditory tube and mastoid cells will be presented to stress the way in which the anatomy lends itself to the spread of infection from the nasopharynx.

The auditory tube communicates with the nasopharynx and the tympanic cavity and serves primarily to equalize the pressure between the two surfaces of the tympanic membrane. Its length is about 36 mm. and its direction is downward, forward, and medialward, forming an angle of about 45 degrees with the horizontal plane.[2] Approximately the upper third of the canal is osseous and the distal

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two thirds cartilaginous and fibrous, the two portions being joined at a narrow isthmus. The diameter of the tube is greatest at the pharyngeal orifice. A small portion of the pharyngeal orifice in the superior aspect remains permanently open; elsewhere the walls are in contact, except during swallowing when they are said to be opened chiefly by the tensor veli palatini muscle.[3]

The anatomy of the auditory tube in infants in particular lends itself to the spread of infection from the nasopharynx to the middle ear. It is much shorter, measuring from 16 to 18 mm. in length, while its breadth is equal to that of the adult.[4] It is also more horizontal and straighter, not having a bend in it at the isthmus as is found in the adult.

The middle ear consists of two parts: the tympanic cavity proper, opposite the tympanic membrane, and an upper smaller attic or epitympanic recess, the greater part of which lies above the level of the membrane. The roof of the attic, the tegmen tympani, consists of a thin plate of bone which separates the tympanic cavity from the middle cranial fossa. Including the attic, the vertical and anterioposterior diameters of the cavity are each about 15 mm. The transverse diameter measures about 6 mm. above and 4 mm. below; opposite the tympanic membrane it is only about 2 mm.[2]

The upper part of the posterior wall of the epitympanic recess leads into an irregular, relatively large cavity called the tympanic antrum. The mastoid air cells communicate with this cavity. These cells vary greatly in number, size and arrangement. Usually they are relatively large near the tympanic antrum and gradually diminish in size as they approach the apex of the mastoid process. The cells in the apex may contain marrow; occasionally they are entirely absent.[2]

At birth the epitympanic recess is present, but the process of pneumatization has just begun and accompanies the growth of the mastoid process. The formation of the mastoid air cells is marked by the fifth year, but development is not completed until puberty.[2]

Morris[3] gives an excellent account of the clinical aspects of the anatomy of the tympanic antrum and mastoid cells:

"Since the floor of the antrum is below the level of the passage communicating with the epitympanic recess, pus in the antrum tends to collect in the mastoid cells. The spread of septic infection from the mastoid region is by several routes: (1) upward, through the thin roof to involve the meninges or through the petrosquamous suture to the superior petrosal sinus; (2) downward, to reach the internal jugular vein or the digastric fossa: (3) forward, to produce

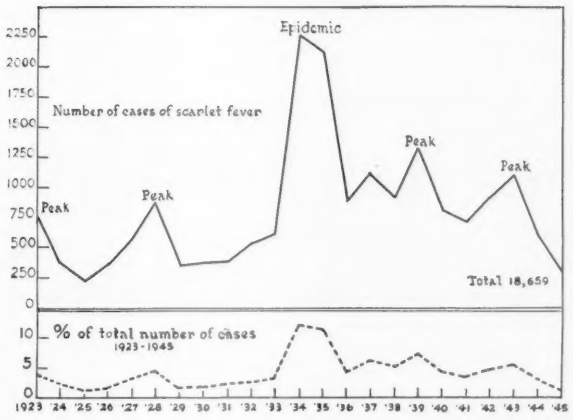
Epidemic
Number of cases of scarlet fever
Peak
Peak
Peak
Peak
Total 18,659
% of total number of cases 1923-1945
1923 '24 '25 '26 '27 '28 '29 '30 '31 '32 '33 '34 '35 '36 '37 '38 '39 '40 '41 '42 '43 '44 '45

Fig. 1.

a subperiosteal abscess just behind the auricle or to perforate and discharge into the external acoustic meatus; (4) backward, to involve the transverse sinus; (5) outward, especially in children, through the thin bone or the unclosed squamosomastoid suture; and (6) inward, to the transverse sinus".

It should be emphasized that there is a continuity of surface between the most distal mastoid air cell in the mastoid process of the temporal bone, the tympanic cavity, the auditory tube and the nasopharynx. At the pharyngeal orifice, the mucous epithelium closely resembles that of the nasopharynx, being a pseudostratified epithelium containing numerous goblet cells. Within the auditory tube itself the existence of glands is generally denied.[5] Here the ciliated epithelium varies from a pseudostratified type near the nasopharynx to a simple columnar type near the tympanic cavity. Within the tympanic cavity the epithelium changes into the simple squamous type; only near the opening of the auditory tube is it of low cuboidal type.

During the years 1923-1945, inclusive, 18,659 patients with scarlet fever were admitted to Southview Isolation Hospital at Milwaukee, Wisconsin. 1,751 or 9.38 per cent of these patients developed otitis media. 141 or 8.05 per cent of the patients who developed otitis media also developed clinical signs and symptoms of mastoiditis. There was one patient who developed spontaneous mastoiditis without apparent signs or symptoms of otitis media. Thus, the total number of patients with manifestations of mastoiditis was

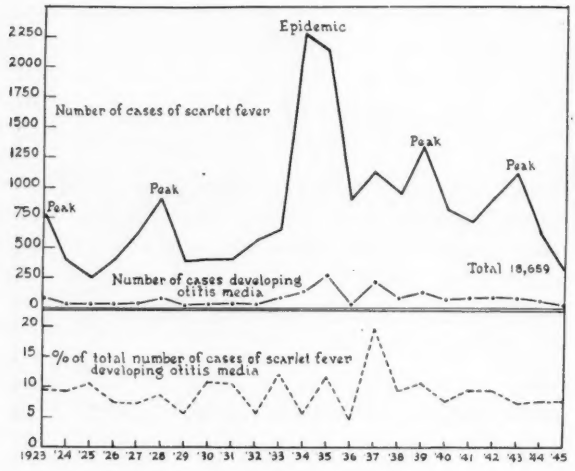

Fig. 2.

142, representing .76 per cent of the total number of cases of scarlet fever.

Figure 1 is a graph representing the number of cases of scarlet fever seen each year and the relative percentage of the total number of cases from 1923 to 1945, inclusive.

Incidence by Years.—From Fig. 1, it can be seen that there was an epidemic of scarlet fever during the two-year period, 1934-1935. During these two years, there were 4,399 patients with scarlet fever admitted, or 23.58 per cent of all the cases of scarlet fever seen during the 23-year period.

There were also small peaks of increased incidence every four to six years. These peaks occurred in the years 1923, 1928, 1934-1935, 1939 and 1943.

In Fig. 2 the graph shows the percentage of patients with scarlet fever developing otitis media each year from 1923 to 1945, inclusive.

From Fig. 2 it may be seen that the incidence of otitis media in scarlet fever has not decreased during the last decade. During the years 1940-1945, inclusive, 4,494 patients with scarlet fever were admitted. 398 of these patients developed otitis media, or

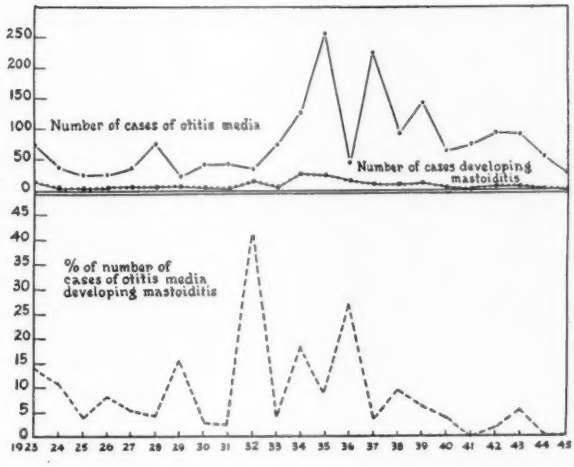

Fig. 3.

8.86 per cent, which does not indicate an appreciable change from the preceding years.

When the graphs pictured in Figs. 1 and 2 are compared, it is obvious that the incidence of scarlet fever and otitis media more often than not did not parallel each other. For example, from Fig. 2, it can be seen that the lowest incidence of otitis media was in 1936 (4.68 per cent) and the highest in 1937 (19.56 per cent); while the number of cases of scarlet fever, 897 in 1936 and 1,125 in 1937, did not vary appreciably from the mean of 1,017 or the average of 811. A further study indicates that in 1937, 221 or 19.65 per cent of the 1,125 patients with scarlet fever were under five years of age, while in 1936, 136 or 15.17 per cent of the 897 patients with scarlet fever were under the age of five years. Since the incidence of otitis media is much greater in the age group under five (Fig. 6), this would in part explain the difference in the incidence of scarlet fever and otitis media during the various years.

The graph in Fig. 3 presents the percentage of patients with otitis media developing "clinical mastoiditis" each year from 1923 to 1945, inclusive. This graph indicates that the incidence of "clinical mastoiditis" has decreased during the present decade. During the 23-year period studied, there were three years in which there were no cases of clinical mastoiditis complicating scarlet fever. These

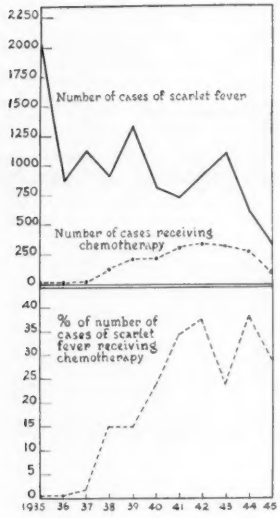

Fig. 4.

three years were 1941, 1944 and 1945—all falling within the present decade.

We hesitate to draw any further conclusions from this group. Here we are dealing with 141 cases, which is a small number compared to the 18,659 cases delineated in Fig. 1 and the 1,751 cases in Fig. 2. Therefore, the graph becomes markedly more sensitive. For example, the year 1932 in Fig. 1 represents 544 cases; in Fig 2 it represents 32 cases, while in Fig. 3 it represents 13 cases.

We prefer to use the term "clinical mastoiditis" because the actual incidence of the involvement of the mastoid cells must in reality be much greater than clinical signs and symptoms indicate. A review of the anatomy would verify this. Certainly the tympanic antrum—the connecting link between the tympanic cavity and the mastoid cells—is involved in all cases of severe otitis media.

Figure 4 shows a graph representing the number of patients with scarlet fever receiving chemotherapy in the form of sulfonamides or penicillin from 1935 to 1945, inclusive.

The decrease in the incidence of mastoiditis in the last decade of this study we attribute to the use of chemotherapy—sulfonamides

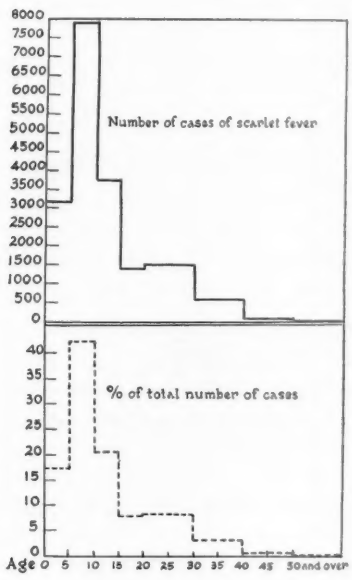

Fig. 5.

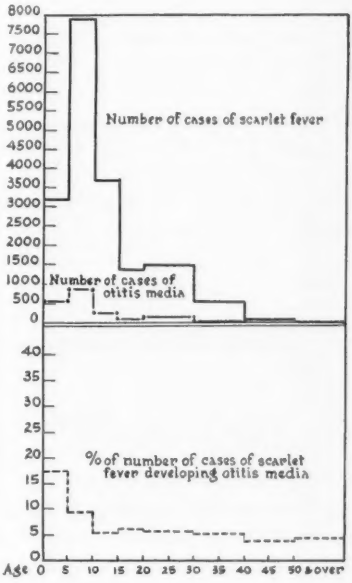
Number of cases of scarlet fever
Number of cases of otitis media
% of number of cases of scarlet fever developing otitis media
Age 0 5 10 15 20 25 30 35 40 45 50 & over

Fig. 6.

and penicillin, which were used whenever there was evidence of a bacteriological septic complication, such as pronounced tonsillitis or pharyngitis, otitis media, purulent sinusitis or bronchopneumonia. 30.66 per cent of the patients received chemotherapy from 1940 to 1945, inclusive.

Chemotherapy is not used routinely in every case of scarlet fever. The dangers of developing drug-resistent strains of bacteria and drug toxicity far outweigh the benefits that can be obtained with its routine prophylactic use. Convalescent serum is indicated to combat severe manifestations caused by the erythrogenic toxin upon which chemotherapy has no beneficial effects.

Incidence by Age.—The incidence of scarlet fever, otitis media and mastoiditis was also analyzed according to the different age groups. Eight different age groups were considered. The first two decades were divided into four five-year periods, the next three decades into three ten-year periods, and the patients over fifty years of age were considered as one group.

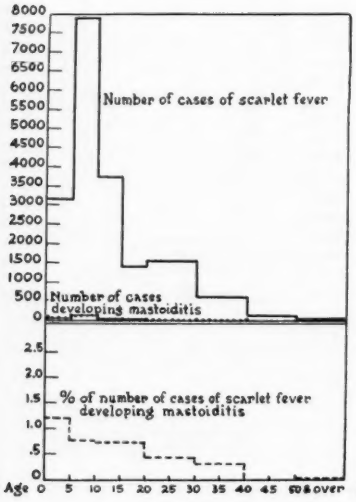

Fig. 7.

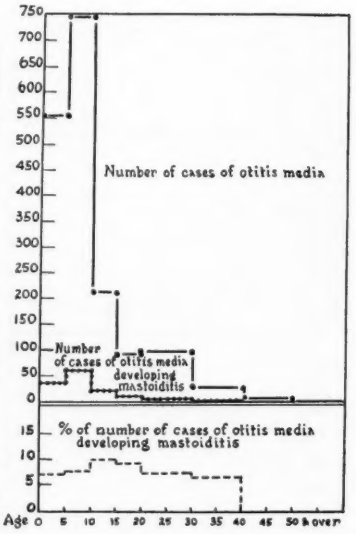

Fig. 8.

The graph in Fig. 5 establishes the greatest incidence of scarlet fever in the age group of 5-9 years, inclusive. In this age group there were 7,940 cases of scarlet fever, or 42.55 per cent of the total number of cases. This was followed by the age group of 10-14 and that under 5 years with an incidence of 20.46 and 17.08 per cent, respectively. In other words, over 80 per cent of the cases of scarlet fever were found in children under 15 years of age. Thereafter there was a marked and steady drop in the incidence of scarlet fever in the older age groups. There appears to be a slight increase in the 20-29 age group over the 15-19 group, but it must be remembered that the latter group represents a five-year age period, while the former represents a ten-year period.

From the graph in Fig. 6 it may be observed that the incidence of otitis media is much greater in the age group under 5 years. In this group, 17.42 per cent of the patients with scarlet fever developed otitis media. This is followed by the age group of 5-9 years with an incidence of 9.43 per cent. This high incidence in the age group under 5 years is to be expected since the anatomy of the auditory tube in infants particularly lends itself to the spread of infection from the nasopharynx.

From Fig. 6, it would appear as though the incidence of otitis media and mastoiditis is higher than would be expected in patients over 50 years of age. However, it must be remembered that the number of patients represented in this group is small—43 acquiring scarlet fever, or a mere .23 per cent of all the patients considered. Thus, when two of these patients developed otitis mdia and one mastoiditis, the incidence of otitis media was brought up to 4.65 per cent and mastoiditis 2.33 per cent. Since the number of cases in this group is so small, we must be careful in making conclusive statements about the incidence of otitis media and mastoiditis in patients over 50 years old.

Figure 7 represents the percentage of patients developing mastoiditis from otitis media. From Fig. 6, it is to be noted that the incidence of mastoiditis quite closely parallels the incidence of otitis media, excluding the age group over 50. Fig. 7, being much more sensitive since it deals with only 8 per cent of the patients represented in Fig. 6, brings out minor differences in the percentage of patients with otitis media developing mastoiditis. This reveals that the patients in the 10-14 age group show a somewhat higher incidence of mastoiditis. In this group there were 3,818 patients developing scarlet fever, 213 otitis media and 23 mastoiditis. Thus, 10.8 per cent of the patients who developed otitis media also developed mastoiditis.

We wish to point out that the patient who developed mastoiditis in the age group over 50 did not have a preceding otitis media, indicating that the spread of infection was in all probability blood borne. This is the only patient in our series who did not have clinical symptomatology of otitis media and further verifies the contention that the occurrence of mastoiditis is almost always by extension from the nasopharynx—whether it be by direct extension, droplet infection or by a submucosal spread.

SUMMARY

1. A brief review of the anatomy of the auditory tube, tympanic cavity and mastoid cells has been presented in an attempt to stress how it lends itself to the spread of infection from the nasopharynx.

2. A statistical analysis of the incidence of scarlet fever and otitis media and mastoiditis complicating scarlet fever by years and age has been presented, embracing the 23-year period, 1923-1945, inclusive.

CONCLUSIONS

1. Out of a series of 18,659 cases of scarlet fever seen over a 23-year period, otitis media developed in 9.38 per cent and mastoiditis developed in .76 per cent.

2. There was an epidemic of scarlet fever during the two-year period, 1934-1935; 23.58 per cent of all the cases of scarlet fever occurring during these two years. There were also small peaks of increased incidence every four to six years; namely, in 1923, 1928, 1934-1935, 1939 and 1943.

3. The incidence of otitis media in scarlet fever has not decreased appreciably during the present decade; 8.86 per cent of the patients developed otitis media during 1940-1945, inclusive.

4. The incidence of mastoiditis complicating scarlet fever has decreased during the present decade. During the 23-year period studied, there were three years in which there were no cases of mastoiditis, 1941, 1944 and 1945, all falling within the present decade. This decrease is attributed to the use of chemotherapy—sulfonamides and penicillin. 30.66 per cent of the patients acquiring scarlet fever received chemotherapy from 1940 to 1945, inclusive.

5. The greatest incidence of scarlet fever was in the age group of 5-9 years; 42.55 per cent of the cases of scarlet fever were in this group. Over 80 per cent of the cases were in patients under 15 years of age.

6. The incidence of otitis media was greatest in the age group under 5 years, 17.42 per cent of these patients developing otitis media.

7. There was a slight increase in the incidence of mastoiditis in the 10-14 year age group; 10.8 per cent of the patients who developed otitis media also developed mastoiditis.

8. Only one of the patients who developed mastoiditis did not have clinical evidence of a preceding otitis media. This was in the age group over 50. This suggests that mastoiditis is almost always due to extension of infection from the nasopharynx.

324 East Wisconsin Avenue.

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XLVI

HEARING AIDS

THE EVALUATION AND PRACTICABILITY OF HEARING AIDS, WITH REVIEW OF RECENT LITERATURE

GORDON BERRY, M.D.

WORCESTER, MASS.

As otologists we are concerned with deafness. We seek to prevent its onset and to control its progress. However, at many points we have failed and the world is abundantly peopled with the hard of hearing. What help does modern science offer? Electrical contrivances for amplifying the spoken voice have been growing in number and efficiency since they were first used half a century ago. As a result of war discoveries, we are enjoying startling advances in these instruments. The surgeon who does fenestration operations becomes enthusiastic, and properly so, if in about half of his selected cases there is a 30 decibel gain. Do we fully realize that the well-fitted hearing aid can give a gain of 40 to 50 decibels and that even those with very little hearing can profit from wearing one?

In exploring this interesting and valuable field we find that hearing aids are serving people of all ages from the preschool child to the extremely aged, and from those with a moderate hearing loss of 30 decibels to those who have a loss of 80 or even 90 decibels.

Among others, Dr. Douglas MacFarlan first satisfies himself that the little three-year-old has some remnant of acoustic power, and then has the mother put a hearing aid on the child and use it in order for her to reach through to its consciousness by the auditory path. It works too; and children who were supposed to be totally deaf are being found to have considerable usable hearing which can be used in the continuing daily contacts about the home.

A few educators in Alexander Graham Bell's day insisted that there was a natural time to learn speech which is the same in the deaf as in those who hear normally, and that if speech is not encour-

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Presented at the Meeting of the American Otological Society, Hot Springs, Va., April 13, 1948.

aged and learned during those early favorable years, there will never again come an opportunity to learn speech so easily or so well. This theory was less enthusiastically held through the next fifty years, but now with powerful and easily applied hearing aids to help, it is attracting more attention in this educational field.

In the young deaf child, the intelligent use of sound amplification is developing a clearer and more rythmic speech than could be secured before this help was available. As a result of the war researches at Harvard under Drs. Harvey Fletcher and Hallowell Davis, the Bell Telephone Laboratories have developed two experimental master hearing aids, to be used with the deaf children at Central Institute and the Clarke School. Microphones, loudspeakers and individual ear pieces permit each who has a sufficient remnant of hearing to talk to the other pupils in the classroom, hear what they say, and appreciate, perhaps for the first time, the corrections in speech articulation that the teacher is making.

The next phase deals with the hard of hearing child in our public schools. A mildly deaf child uses lip reading and takes a seat near the teacher. The harder of hearing child wears his hearing aid and follows the teacher with little or no trouble.

As high school and college are reached, those who have early been deafened have arrived at an orderly and established means of meeting and overcoming this handicap. But the progressive forms of deafness that first appear in young adult life may offer educational and psychological repercussions, if they are not energetically and sympathetically taken care of. Here the otologist can do much to adjust both parent and child in the early acceptance of the hearing aid in advance of when it becomes urgently needed. Such acceptance saves the child many a failure in the classroom and many a heartache in his or her social contacts.

Finally in adult life the hearing aid is becoming so easy to wear and so efficient when used that many more are using one. One writer estimates that 800,000 are now in use. In 1943, only 110,000 hearing aids were purchased from the entire industry. In 1946 or three years later, 225,000 were purchased, or more than double. The average age of these purchasers is 55 years, and their number is increasing. A recent survey found sixteen and a quarter million people in the United States over fifty years of age in 1920, and thirty-one million in 1947.

In this situation it may be argued that lip reading no longer becomes necessary. A recent study by Dr. Clarence V. Hudgins of

the Harvard research group offers significant data. He tested the responses to spoken words on 26 children at the Clarke School for the Deaf, first with lip reading alone, then with the hearing aid alone, then with both used together. The average hearing impairment for these children showed a 78-decibel loss. The score for lip reading alone was 43%; for a hearing aid alone, 23%; when both lip reading and the hearing aid were used, 65%. This was in so-called deaf children whose impairment was great and whose school training had been emphasizing lip reading. The lesson is that we encourage our patients to use both the hearing aid and lip reading, not either one alone. Having touched on the many who are profitting by the use of the hearing aid, let us now turn our attention to the modern application of its use and then to the recent changes in the instrument itself. The important procedure shared by all the army and veterans' programs was the constructive manner in which, over a four to six weeks' period, the veteran was trained in the use of a hearing aid; taught the supporting art of lip reading; given classroom work where he listened to instrumental and vocal music, to the spoken voice with and without background noises, to conversations over the telephone; and finally, encouraged in its use in general conversations, active sports, social gatherings and dancing. After the confidence gained in such training more than 90 per cent of the discharged navy veterans continue to use faithfully the hearing aid given them. This is a very different story from that of the many discouraged civilians who put their hearing aids up on the closet shelf after only a cursory trial. Auricular training greatly helps early hearing aid users.

In the program for civilian hearing aid clinics the Academy of Ophthalmology and Otolaryngology urges that each state have at least one hearing clinic or center where careful otological reviews and tests can be made and a thorough fitting for a hearing aid be made, patterned somewhat after the Army and Navy plan. Also many leagues for the hard of hearing are introducing hearing aid service into their program.

The Harvard report found "that regardless of the nature of their particular defect, most patients hear best with an instrument which amplifies all frequencies uniformly, or with moderate emphasis on the higher frequencies." This is the principle of "high fidelity" as against the commonly accepted principle of "selective amplification" which tries to so amplify the valleys of the patient's audiogram as to make an over-all flat curve as near the normal as possible. As future hearing aid manufacture will doubtless be strongly influenced by the new concept advanced by the Harvard Studies on Hearing

Aids it will be helpful to record here the major specifications recommended.

> "Frequency Response and Range.—Uniform, i.e., without marked resonant peaks or valleys, from 300 to 4,000 c.p.s. Sharp cutoffs below and above this range are desirable. The one best frequency characteristic is a moderate high-tone emphasis of from 4 to 6 db. per octave over this range.
>
> "Tone control.—Preferably by compression amplification; alternatively by simple symmetrical peak clipping.
>
> "Maximum Output.—Semipermanent adjustment, or provision of separate models, at 114, 120, 126, or 132 db. re 0.0002 dyne/cm. maximum instantaneous acoustic pressure.
>
> "Maximum Acoustic Gain.—Separate models would probably be desirable, the lowest powered to have at least 40 db. acoustic gain available, and the highest powered a maximum of 80 db.
>
> "Gain Control.—Smoothly graded or in small steps on an approximately logarithmic scale over a 40 db. range.
>
> "Intrinsic Noise.—Must not mask speech delivered to the instrument at a sound-pressure level of 30 db."

Thus we find that the fitting of hearing aids should be based primarily on the maximum acoustic output (or ceiling) required and bearable by the user, with little effort to conform to the individual audiogram curve.

The last phase of my investigations for this paper concerns the generous replies from different manufacturers to whom I wrote for information as to recent advances in their instruments.

The first over-all comment is the increase in acoustical gain. The consultants on Hearing Aids of the American Medical Association laid down the requirement only a few years ago that a hearing aid must be able to give a minimum acoustic gain of 30 decibels. Now 45 decibels is a modest claim. One reliable manufacturer says his latest model delivers an average gain of 55 decibels in the speech range, and that a patient with a 95-decibel loss "can hear comfortably" with this hearing aid. Another well-known make claims a 65-decibel acoustic gain with the full power on. For 17 of the recognized instruments now on the market, one report showed the lowest maximum acoustic gain to be 44 decibels, the highest 74 db. and the average 62.6 db.

Every wearer wishes to have the size and the weight reduced. Now, instead of large cumbersome batteries worn in a separate case, the batteries and the transmitter are all included in one unit and,

combined, the size is the same or smaller than the transmitter alone two years ago.

A recent refinement takes the ear receiver out of the ear and places it below the collar under the clothing. A small flesh-colored plastic tube leads from the receiver up to the ear, ending in a small funnel which is hidden in the folds of the ear or even in the hair. This tube is so small that it does not carry sound waves to the receiver in sufficient quantity to make this device truly efficient.

Not only are the cases lighter and better looking, but they are being made of stronger plastic. One company claims you can put its case on the floor and step on it without injury. Ethyl cellulose as a plastic is reported as having a low coefficient of friction and being a good dampener of clothing sounds. Another refinement is a recession in the vent through which the sound enters, thus cutting down clothing rub. Also the wire connections and the amplifier have been mounted in soft butyl rubber, further reducing noises.

Perhaps the outstanding changes concern the battery, the ear receiver, the radio tube and the circuit wiring. The use of mercury batteries for walkie-talkies during the war gave the impetus to their use in hearing aids. They are being made smaller and yet smaller and are proving very effective for the low voltage A battery. They maintain a more even current output and voltage capacity, but drop off more suddenly when exhausted than the carbon type battery does. This carbon cell battery has also been made much smaller and longer lived through a new "layer built" construction and an improvement in the pastelike "mix" which serves as the chemical agent. The new small batteries are not as economical as the older heavy ones. Battery cost estimates listed for 17 of the better known instruments range from 0.62 to 3.0 cents an hour with an average hourly cost of 1.24 cents.

The ear receiver plays an important part in the fitting of a hearing aid. Many companies use three types. In general, one type flattens the peak, one cuts down the low tones and one cuts down the high tones. Also the microphone carries a tone control, successively dampening the low frequencies. Then, with a higher or lower voltage in the batteries, the desired amplifications can be secured. Different companies have different methods for arriving at the best fitting. In general, the aim is first to secure the needed amplification, second, to limit the maximum output to within the user's tolerance, third, to use that ear receiver and the interior combinations which will tilt the frequency response to the user's need. Such adjustments

avoid the distortions of the earlier hearing aids and make it possible to help hearing losses as great as 90 decibels.

The best ear molds were made of plaster of Paris. They are exact but tend either to break at the external auditory meatus, or lock in and make it hard to remove them. Plastic engineers have been able to evolve a soft elastic plastic which is inserted more easily, sets more quickly, and is removed without difficulty. Many hearing aid agents make their own ear molds with safety and dispatch, but there still remains the possible presence of a plug of wax in the ear canal to militate against a good fitting unless it is first removed.

While most of the hearing aids use crystal microphones, the majority of the ear receivers are of the magnetic type. This latter has been made possible by improvements in magnetic materials and in mechanical design. The crystal used is Rochelle salt which unfortunately is sensitive to moisture and temperature changes. Reducing the size of a crystal diaphragm makes it increasingly difficult to reproduce low frequencies, so crystals are used more easily in the larger diaphragm of the microphone than in the smaller one of the ear receiver.

One manufacturer uses a magnetic microphone. One advantage of this type is its capacity to pick up telephone and radio conversations. The introduction of a small inductive pickup coil into the unit permits the direct pick-up of audio magnetic waves surrounding the telephone receiver. A small coil can be similarly attached to a radio set. This inductive coil picks up not sound waves but magnetic waves, direct, and eliminates much of the usual distortion. Another feature places the sound vent in the side of the case, lessening clothing friction noises.

Improvements in radio tubes are too intricate for the writer to follow. We understood the tremendous change from the original large radio tube to the small midget tubes; yet more refinements are now possible. These tiny tubes have ever-increasing power capacity and a lengthened operating life. There is also a reduction of the microphonic noise caused by the vibration of the heated tube filaments which formerly caused bell-like tones in the receiver. The power yield is greater, while the B battery voltage needed to obtain that power is less. This permits a smaller size B battery. The current drawn from the 1½ volt A battery has been reduced, resulting in either increased battery life or a smaller size as the manufacturer or user may choose. Other items in the amplifier are the improved resisters and condensers in the electric circuit and the better insula-

tion and better shielding which improve the stability of the instrument.

An innovation which two hearing aid companies have accepted enthusiastically and several others are planning soon to use, is the so-called printed circuit. This is another invention coming out of the war, which advanced in so many ways the science of acoustics.

The story starts with the development of the tiny generator-powered radio proximity fuse for the U. S. Army's mortar shell. This is the shell that exploded when coming within a predetermined distance of its objective, such as an enemy airplane. The fuse had to be small, about the size of an ordinary radio tube. Completely self-contained in the fuse were the radio-transmitter-receiver, control circuits, safety devices and a turbo-generator power supply. It was placed at the front end of the shell and had to withstand a pressure 10,000 times that of gravity, together with the great heat from air friction while in flight. The electrical control circuit to meet these rugged requirements and at the same time to be free of short circuiting from moisture, was found in the tiny printed circuit. In modified form it is now used in some hearing aids. The base material is steatite, a dense, porcelain-like ceramic material of the hardness of sapphire. This is pressed or cast, then dried and fired at 2400° F. to shrink to a hard flat thin plate about 1¼ to 2½ inches in size.

Then a stencil is prepared, usually by a photographic method. Through this stencil a silver paint is printed onto the ceramic plate. This is heated in a furnace to 1400° F. bonding the silver to the ceramic with a tensile strength of 3000 pounds. This silver will serve as the conducting wires in the circuit.

Resisting units are introduced by the careful spraying on of a resistance paint with a known resisting co-efficient. This is fixed in an oven at 300° F. and then covered with resin to protect it against humidity. Third, ceramic disc capacitaters are molded and attached. Finally tiny radio tubes and external leads are soldered into place. The result makes a solid small unbreakable unit, impervious to temperature and moisture changes, uniform in construction, eliminating the time consuming and detailed hand work of assembling 173 small parts and the soldering of 65 joints. Different sizes in the plate and different arranging of the printed circuit are readily made to suit the particular design and need.

Repair of a unit simply means the installation of a fresh printed circuit and the return of the damaged one for factory repair. When

detailed requirements for individual hearing aid manufacturers have been ironed out, it would seem highly probable that this printed circuit will be an integral part of most hearing aid instruments.

SUMMARY

1. This review finds a sharp advance in the production and use of hearing aids since the war.

2. They are being used to advantage in the very young to gain better articulation and rhythm in those early years when the child normally learns speech.

3. In the school years as well as in later life, it is pointed out that lip reading and hearing aids help greatly but that neither alone helps as much as both used together.

4. The war program has taught us the value of auricular training in becoming adjusted to the use of hearing aids.

5. The Harvard Studies during the war have questioned the value of "selective amplification" which tries to match the audiogram curve and recommends a fitting on the principle of "high fidelity." This places a ceiling on the acoustic output ranging from a noise level of 115 db. up to as high as 130 db. when the individual's tolerance will permit it. To avoid overemphasis of the loud vowel sounds, the low tones are suppressed, while the curve is tilted upward 3 to 6 db. per octave to accentuate the consonant sounds, which range above 1000 frequencies. This will lessen distortion and make for clarity.

6. Reports from the engineers of the different companies point out many advances. (a) Batteries are of two types, carbon and mercury, and improvements in materials and assembly have made them much smaller and more efficient. (b) This has permitted a drastic change in the size of the transmitter which is now put in a single case. (c) Ethyl cellulose for the case, butyl rubber mountings for insulation, smaller and moisture-proof cords, all make for reductions in clothing-rub noises that so trouble the user. Even the vent in the plastic case has been adjusted to further stop these friction noises. (d) The midget vacuum tubes have been made still smaller, more powerful and free of objectionable resistance noise. (e) The ear receivers are smaller and more efficient. Magnetic receivers have been so improved that many manufacturers use the crystal diaphragm only in the transmitter. One firm has finally found what it considers a more efficient magnetic diaphragm for the transmitter as

well. (f) Plastics for making ear molds are so improved that hearing aid agents can safely make them, provided any obstructing wax has first been removed. (g) Elaborate devices are being designed to assist in the selective fitting of the hearing aid. These will be simplified and made more efficient as the significance of the Harvard Studies are better understood. (h) Another war product is the printed circuit which is being adapted to use in the hearing aid. It will save much skilled labor in small part assembly, and makes for ruggedness, protection against moisture, and simplicity. Some hearing aids are using it with enthusiasm, while others are waiting for electronic engineers in cooperation with the Bureau of Standards to correct the few faults they think persist. These many improvements are probably but a forecast of other and better refinements yet to come. There is healthy and keen competition and continuing advances seem assured.

CONCLUSION

In conclusion, hearing aids have assumed an important part in otology. Most of us cannot be electrical or acoustic experts, but we can understand the general mechanism, what acoustic amplification can accomplish and how this acoustic help can wisely be applied to our patients. This is a relatively new field, one that the next generation will understand better than we do. Happily the acoustic experts are charitable and will cooperate with us in helping our patients. On them will rest the burden and the privilege of devising yet more wonderful instruments for the alleviation of deafness which has advanced beyond our skill to remedy. Ours is the continuing task of discovering the means to prevent that deafness.

Much of the source material for this review was secured from the current otological publications and the Volta Review, and from "Opportunity and the Deaf Child" (Irene and Alexander Ewing, University of London Press), and "Hearing and Deafness" (Hallowell Davis, Murray Hill Books, Inc.). For information regarding various details of hearing aid engineering I am indebted in turn to William E. Snodgrass of the Western Electric Company, David H. Barnow of the Beltone Hearing Aid Company, L. A. Watson of the Maico Company, Fred W. Kranz of the Sonotone Corporation, S. F. Lybarger of E. A. Myers and Sons, Grover C. Coil of the Allen-Howe Electronics Corporation, W. S. Parsons of the Globe Union Co., and Cledo Brunetti of the National Bureau of Standards.

36 PLEASANT STREET.

Clinical Notes

XLVII

MULTIPLE FRACTURES OF THE FACE AND PRIMARY SHOCK, COMPLICATED BY CONGENITAL CLEFT PALATE

REPORT OF A CASE

MERTON N. FLANDERS, M.D.

WATERVILLE, MAINE

Adams[1] has brought our attention to a method of quickly securing complete immobilization of facial fractures. He contends that the numerous types of extra-oral appliances, including those attached to plaster headcaps, for immobilizing fractures of the maxilla and other facial bones, are complicated; their preparation and application are time-consuming; they are cumbersome and uncomfortable for the patient; and they require close watching and repeated adjustments on the part of the surgeon. The fact that everyone treating these fractures has a different type of extra-oral appliance suggests that this method is not entirely satisfactory.

Internal wiring fixation, as described by Adams, was used on the patient described in the following case report.

Miss A. C., 18 years old, was received at the hospital at 3:25 a. m., June 22, 1946, about five hours after a serious automobile accident. She was in primary shock from trauma, hemorrhage and a long ambulance ride. The soft tissues of the face were considerably swollen and the maxillae appeared depressed. There was active bleeding into the nasopharynx, and the tongue slipped back into the throat, partially obstructing respiration. Swallowing was either impossible or so difficult and painful that she did not attempt it, and to keep her airway clear she blew the collecting blood from her

From the Thayer Hospital, Waterville, Maine.

Presented before the New England Oto-Laryngological Society, Boston, Mass., November 19, 1947.

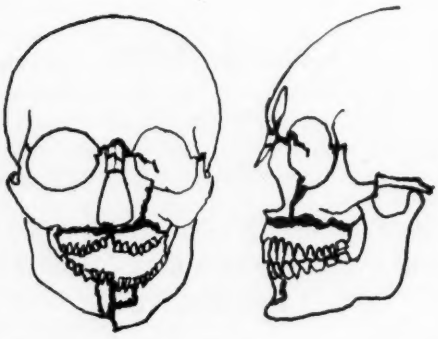

Fig. 1.—Diagramatic reproduction of x-ray pictures showing fractures.

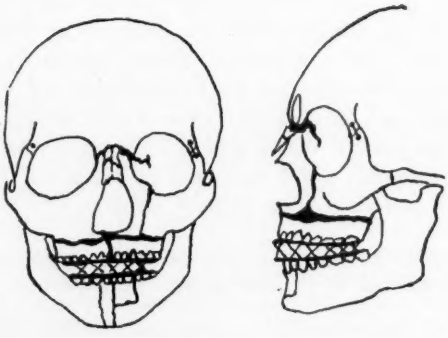

Fig. 2.—The halves of the maxilla have been pulled down and forward and a half bar (arch band) loosely wired to the upper teeth. A half bar was wired to the lower teeth to align the fragments of the lower jaw. Securing the best occlusion possible, the jaws were wired together and all wires were tightened.

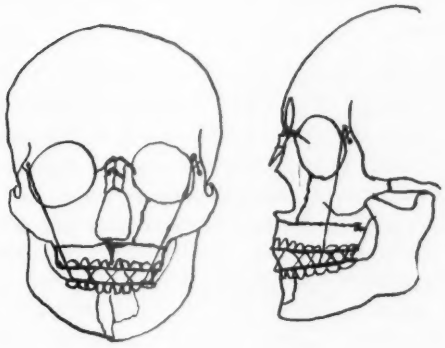

Fig. 3.—An incision was made over each frontozygomatic suture. On the left side it was necessary to wire the two bones together, because of the amount of separation and dislocation. A small hole was bored in each frontal bone close to the frontozygomatic suture. A small steel wire was threaded through the hole and then both cut ends of the wire were threaded on a large straight needle. The needle was passed into the small incision already made and through the deep tissues of the face into the upper buccal sulcus about opposite to the second molar teeth. This procedure was carried out on the opposite side of the face. The free ends of the wire were then passed around the lower arch band and twisted up tightly until the reduced fractures were impacted.

mouth. A scar could be seen extending vertically from the lower border of the right external naris down through the lip. The patient did not know whether she had had a cleft palate or not. The right upper central incisor was absent and only a small space intervened between the left central incisor and the right lateral incisor. A fracture at this point was obvious because of the angulation at this point of the two halves of the maxilla. Other injuries were a laceration of an elbow and one ragged laceration of a flexor tendon of the left hand.

Treatment for the shock was instituted at once and gas gangrene and tetanus antiserum administered. The wound of the flexor tendon of the hand was irrigated and debrided soon after admission by the orthopedic consultant. Sterile dressings were put on until such time as she would be in condition for operation. A Levine tube was passed through the nose and into the stomach to supply fluids and

nutrition. Penicillin therapy was started and two blood transfusions were given.

Two days later roentgenograms were made. The x-ray report was: "Thorax and lungs normal. Extremities normal. Skull and cervical spine normal. Face: Cleft palate with separation of the halves of the maxilla through the floor of the right nasal chamber. Comminuted fractures of the nasal bones which are raised at the nasofrontal suture. The fractures extend into the ethmoids. Fractures of both malar bones. On the left there is a comminution of the floor of the orbit. The zygomatic-frontal sutures are separated and displaced, particularly on the left. Antral walls are fractured. Both antra are opaque. There is a fracture of the symphysis of the mandible with comminuted fractures of the alveolar process."

Three days after admission an elective tracheotomy was done so that the airway might be kept clear of blood or emesis that might complicate the reduction of the facial fractures and the intermaxillary wiring under ether anesthesia. On the following day, the patient was given a basal anesthesia of avertin, and light ether was administered through the tracheotomy tube.

The facial fractures were reduced and an opening was made into the left antrum through the canine fossa. The lumen was found greatly decreased in size and filled with a blood clot. A rubber tissue drain was sewed in place.

On the following day the Levine tube was removed because the patient was able to swallow fluid. Several days later the orthopedic surgeon repaired the left hand and requested the continuance of penicillin. On July 5 the tracheotomy tube was removed. Penicillin therapy was discontinued by the orthopedic surgeon on August 6, the patient having received a total of about 3,000,000 units in 45 days. On August 7, under local anesthesia, the wires were removed after being in place for 42 days.

When removing the internal facial wires, it is advantageous to reopen the incisions overlying the frontozygomatic suture lines. The wire is then exposed where it passes through the drill hole in the bone. Both strands of the wire are cut in the mouth, care being taken that no hooks or catches are left on the cut ends. The wire is pulled up through the deep tissue of the face, emerging from the frontozygomatic incisions. No infection of the face has been known to follow this procedure.

At the time of discharge, several days later, the facial contours had been restored and the patient was able to chew solid food. Six months after discharge, the upper central teeth, which were crooked and not in occlusion with the lower teeth, were extracted. A partial denture was made to improve function and appearance.

THAYER HOSPITAL.

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XLVIII

FIBROSARCOMA IN A FOREIGN BODY GIANT CELL TUMOR OF THE LARYNX

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AND

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Extensive studies of the incidence of sarcoma of the larynx have been made covering a 30-year period by Havens.[1] In his series the tumors were definitely of the malignant type and in a few instances tumor giant cells with multiple irregular nuclei were seen but there is no reference to any tumors showing the characteristics of a benign foreign body type and a fibrosarcoma in the same tumor. In the literature available to us, we have been unable to find any report of a similar case.

In the past there has been indiscriminate usage of the term giant cell sarcoma. This has resulted in some confusion as to the true nature of the tumor. Some authors, Meyerding[2] in particular, have suggested that the terms "benign giant cell sarcoma" and "malignant giant cell sarcoma" be used, in the belief that such designation would result in a definite differentiation of the benign and malignant types.

Pathological giant cells may be divided into foreign body cells and true tumor cells The foreign body giant cells are derived from macrophages of reticulo-endothelial origin, which by fusion form large multinucleated cells. The nuclei are placed peripherally. They occur in granulation tissue and are specific reactions of the endothelium to inflammation. The true tumor giant cells, on the other hand, are altered neoplastic cells and may occur in any type of malignant tumor, whether carcinoma or sarcoma. They are the result of too rapid multiplication of actively growing tumor cells. The nuclei are usually central. When such cells occur in considerable numbers in a tumor, they are a sign of malignancy.

The foreign body giant cell places the tumor in a clinically benign class and the term "benign giant cell sarcoma" is applied to it.

The sarcomata characterized by the presence of true giant cells are markedly malignant and the term "malignant giant cell tumor" is used.

Sarcoma of the larynx is comparatively rare. In the series reported by Havens, sarcoma of the larynx occurred in the ratio of 1:100 to squamous cell carcinoma. The ratio of all other types of malignant disease of the larynx to squamous cell epithelioma is 1:44.

The symptoms vary, depending on the size and location of the tumor. Hoarseness is most likely to be the first symptom. Dyspnea seldom occurs but may develop rapidly because of a sudden increase in the rate of growth or an inflammatory reaction which causes a swelling of the tumor with partial obstruction.

Sarcomas of the larynx are a greyish purple color, lobulated, and may be rather extensive in size but still not show a great deal of infiltration of the vocal cord. Many are pedunculated. They do not tend to ulcerate, which is in contrast to the early ulceration of carcinoma. The tumor presents a rather benign appearance; however, one must be suspicious of any tumor in which there is a rapid increase in size. Positive diagnosis is possible only on microscopic examination of the tissue from the tumor. Fresh frozen sections should be made at the time of the biopsy; in doubtful cases, careful study of fixed frozen sections is the method of choice.

The treatment of sarcoma of the larynx has been well summarized by Figi[3] who wrote:

> "The treatment of sarcoma of the larynx depends on its size, situation and activity. Since many of these tumors are pedunculated, show less tendency to infiltrate the laryngeal structures and metastasize later than do carcinomas of the larynx, they may remain operable for a considerably longer period. Many of them are quite inactive and in an early stage may be removed by direct, indirect or suspension laryngoscopy. If such removal is followed by electrocoagulation of the attachment of the tumor, the prognosis will be good in many cases."

REPORT OF A CASE

Mr. R. McA, aged 76, a retired railroad engineer, was examined November 3, 1947. At that time he complained of a chronic hoarseness. About ten years ago he gradually became hoarse. This became more severe for a time and then remained about the same until three months before examination. Since that time the hoarseness rapidly increased until by the time we saw him he was unable to speak above a whisper. At no time did he complain of a sore throat or pain in the larynx. Breathing had not been impaired but during the past three months there was a slight shortness of breath. His

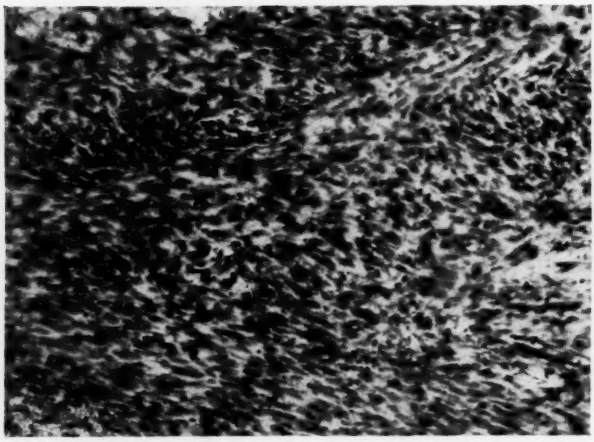

Fig. 1.—Photomicrograph of tumor tissue showing grade II fibrosarcoma. X 165.

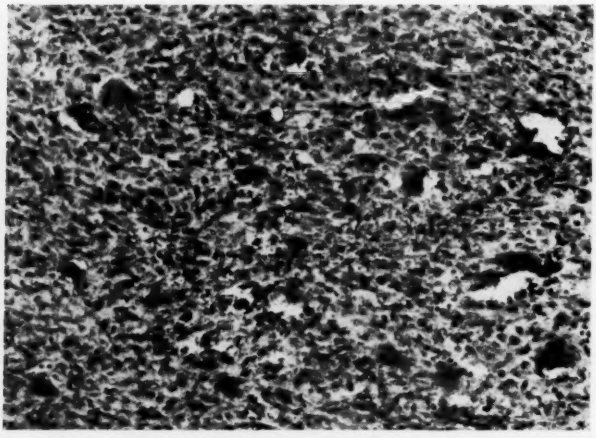

Fig. 2.—Photomicrograph of a different field from the same block of tumor tissue as Fig. 1, showing foreign body giant cell tumor. X 165.

general health had been fair. During the past year he had been treated for hypertension. Blood pressure was 190/110. The remainder of the physical examination was essentially negative.

On examination there was a large tumor mass involving the middle portion of the left vocal cord. The tumor was irregular in outline and had a smooth lobulated appearance. The base was small and there was no infiltration of the surrounding tissues. Under direct laryngoscopy a piece of tissue was removed for biopsy. This was diagnosed as sarcoma with many areas of malignant cells suggesting grade IV squamous cell carcinoma. Laryngofissure was done on November 11 under local anesthesia.

At operation a large, smooth, lobulated tumor mass was found involving the middle third of the left vocal cord. The tumor measured about 1 cm. in diameter and was attached by a narrow base. The left vocal cord was removed. There was considerable bleeding which was controlled by diathermy. The patient made an uneventful recovery and left the hospital on the sixth postoperative day. One week later he was readmitted to the hospital with an acute urinary retention. An indwelling catheter was inserted and the acute symptoms relieved. On November 19 a transurethral resection was done. Recovery was uneventful and he left the hospital on the tenth postoperative day.

The vocal cord and tumor were sent to Dr. A. C. Broders of the Mayo Clinic. Fixed frozen sections of the tumor were made and diagnosed as grade II fibrosarcoma in a foreign body giant cell tumor.

SUMMARY

1. In the literature available to us, we have been unable to find a report of a similar fibrosarcoma in a foreign body giant cell tumor.

2. Sarcoma of the larynx is quite rare, occurring in the ratio of 1:100 to squamous cell carcinoma.

3. Treatment of choice, in the smaller lesions, is removal through direct laryngoscopy. Larger lesions are removed by laryngofissure.

WESTERN MONTANA CLINIC
DIXON BUILDING.

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XLIX

HEMANGIOMA OF THE LATERAL PHARYNGEAL BAND

REPORT OF A CASE

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The lesion concerned in this study is of interest because of its location, the unusual clinical symptoms it produced, the rarity of occurrence of such a tumor in the pharynx and the fact that the pathologic nature of the lesion was not identified until after the tumor had been removed.

"Lateral pharyngeal band" or simply "lateral band" is the term commonly employed to identify the lymphoid structure occurring in the lateral pharyngeal wall posterior to the posterior tonsillar pillar. In standard textbooks[1, 2] this structure has not been given the importance it deserves clinically. It consists of an accumulation of superficial lymphoid nodules.[3] It is common among persons in whom there is a tendency toward lymphoid hyperplasia. As such, the lateral band is a component of Waldeyer's tonsillar ring. Each band extends up to, and is continuous with, the salpingopharyngeal fold; inferiorly the band extends to the level of the inferior pole of the faucial tonsil. The bands usually are red and swollen when one of the common infections of the upper part of the respiratory tract is present. So-called lateral pharyngitis refers to an inflammation localized to the lateral band.

REPORT OF A CASE

A married Filipino business man, 45 years old, first registered at the Mayo Clinic on February 9, 1948. He had been referred to the clinic because of a "nasopharyngeal tumor" which "appeared malignant." Symptomatically, his chief complaint was recurrent paroxysms of coughing of one year's duration.

The recurrent cough was associated with a feeling of "sore throat." The patient had done nothing about this until two months prior to his registration, when, for three successive nights, he had

been unable to sleep because of repeated coughing. He therefore had consulted a physician, who carried out electrocoagulation of the "peanut-like" lesion of the left side of the pharynx. Because of subsequent pain referred to the left ear, the patient sought the advice of a physician friend, who at first suggested that the pain in the ear occurred as a result of use of the cautery. When the patient was examined, however, this physician observed a "nasopharyngeal tumor." Biopsy was performed and, because of much swelling of the patient's neck ("like mumps"), penicillin was administered every three hours for two days thereafter. Because of the lack of technical facilities in the Philippine Islands as a result of the war, pathologic examination of the specimen removed for biopsy required about five weeks. Finally, the specimen was reported as "suspicious" of cancer; it was said probably to be an epithelioma. The patient then was referred to the clinic by his physicians.

The patient's history revealed that "tonsillar abscesses" had been present repeatedly until the performance of tonsillectomy in 1938. He had had malaria in 1928, and typhus and bacillary dysentery in 1930. Otherwise, the history was irrelevant.

Physical examination revealed a short, well-developed, well-nourished, genial, cultured Filipino male who did not appear to be ill but who was agitated to the point of phobia by a foreboding as to the presence of cancer.

Positive results of the otolaryngological examination were confined to the left lateral oropharyngeal wall. The lateral pharyngeal band was very red, edematous and thickened. The lesion was not circumscribed, but blended into the adjacent tissues. Examination of the nasopharynx by mirror and with the nasopharyngoscope disclosed the rim of the eustachian tube to be situated above the lesion and uninvolved. This could be verified also by observation through the left inferior meatus of the nose. Inferiorly, the lesion appeared to end at the level of the lower pole of the tonsillar fossa.

The lesion was very discrete. In general, it did not appear to be malignant. It seemed to vary in size at different times. In an attempt to make a clinical evaluation, the patient was examined by a number of otolaryngologists. On two occasions a very competent otolaryngologist felt that there was no abnormality at all, and on a third occasion he noted only a "fullness on the left," illustrating the apparently insignificant appearance of the lesion.

The vocal cords were of normal appearance, moving and approximating normally. Masses were not palpable in the cervical region.

A complete general physical examination disclosed no organic changes of significance. Systolic blood pressure was 118, and diastolic, 80, expressed in millimeters of mercury. The eyes were normal, without evidence of Horner's syndrome. Dermatologists noted verrucae seniles on the face and cutaneous tags on the neck. Results of a complete blood count, including erythrocytes, platelets, hemoglobin, and a differential count, were normal except that eosinophils amounted to eight per cent. With the exception of Trichuris trichiura, found on one occasion, the stools did not contain ova or parasites, and the presence of the whipworm probably accounted for the eosinophilia. Plasmodia could not be detected in a thick smear. Results of urinalysis and of routine serologic tests for syphilis were normal. Roentgenograms of the sinuses, neck, thorax, colon and terminal part of the ileum showed these structures to be normal.

Two fixed sections of the specimen taken for biopsy in the Philippine Islands were brought by the patient. The opinion of Dr. J. R. McDonald was that these slides showed "necrotic inflammatory tissue."

Squamous cell epithelioma, grade 4 (shown by New[4] to be the true nature of the so-called lympho-epithelioma or Schmincke tumor), was considered in the differential diagnosis, but since there was nothing to suggest a malignant process, a preoperative diagnosis of hypertrophy of the left lateral pharyngeal band was made.

At operation, topical and infiltrative anesthesia proved eminently satisfactory. Premedication consisted of the administration of 0.1 gm. (1½ grains) pentobarbital sodium the night before operation; the same amout of pentobarbital sodium plus 0.016 gm. (¼ grain) of morphine sulfate and 0.00043 gm. (1/150 grain) of atropine sulfate was administered preoperatively.

The mass was dissected with scissors and the base was burned with surgical diathermy. There was surprisingly little bleeding. Since there was no gross evidence of a neoplasm, a fixed section was prepared. The pathologic report showed that microscopically the tumor showed irregular, sometimes anastomosing, blood spaces varying markedly in size as well as in shape. These spaces were lined by a single well-defined layer of endothelial cells, and for the most part were separated from each other only by narrow septa of fibrous tissue. In the peripheral portions of the tumor, there was a marked increase in the amount of connective tissue stroma, with wider separation of the blood spaces. The borders of the tumor were poorly defined, with irregular blood spaces extending in some areas up to

the covering epithelium and in other areas surrounding and penetrating between the lobules of the deep seromucinous glands. Dr. E. M. Parkhill made the diagnosis of hemangioma.

The mucosa healed rapidly. When the patient was examined one week postoperatively, it appeared that the hemangioma had been completely removed. Re-examination of the vocal cords revealed normal function, and there was no manifestation of Horner's syndrome. Symptomatically, there was only the mildest occasional feeling of soreness.

Dr. R. E. Fricke carried out prophylactic irradiation with 4,800 milligram hours of radium, applied externally.

Hemangioma is one of the rarest benign tumors which occur in the pharynx.[5, 6] Of 357 patients with tumors of the tonsil and pharynx seen at the Mayo Clinic in the period 1917 to 1930, inclusive, and reported in the classic study by New and Childrey,[7] 63 (17.6 per cent) had benign lesions and only 2 had hemangiomas. The two who had hemangiomas were a boy seven years old and a girl ten years old. In another series[8] of 99 nonmalignant tumors of the oral and pharyngeal cavities, of which 34 were situated in the pharynx, only 5 were hemangiomas. Eggston and Wolff[9] tabulated 379 instances of neoplastic disease of the palate, tonsil and nasopharynx seen at the Manhattan Eye, Ear and Throat Hospital for a 14-year period, but they listed only 4 hemangiomas. Hallberg[10] reported only 8 in a total of 236 benign tumors of the soft palate. In all 8 of his cases the hemangiomas either had been present since birth or had appeared shortly thereafter.

A rather extensive search of the literature for similar cases revealed only one in which the lesion was in such a definitely circumscribed location. Lichtwitz[11] in 1895 described the lesion as a dark brown, occasionally bluish tumor situated behind the left tonsil, 3.5 cm. long and 1.5 cm. wide, running vertically up to within 0.5 cm. of the rim of the eustachian tube, and inferiorly to the level of the lower pole of the faucial tonsil.

In other comparable cases the lesions described usually are much more extensive, such as that of Wolfenden[12] and the very widespread cavernous hemangioma of the hard and soft palates, anterior and posterior pillars of the pharynx, with extension to the larynx, described by Woodward.[13]

Many other types of tumors, benign and malignant, occurring on the lateral nasopharyngeal wall exclusive of the tonsil, are re-

ported, and the possible presence of examples of them must necessarily be considered in the differential diagnosis. Invariably, they were of such extent as to be obvious on inspection. They include hyperplastic inflammatory tissue, fibromyxoma,[6] lipoma,[14] neurofibroma (schwannoma)[15-18]—the first of which was reported by Figi[19] in 1933—ganglion cell neuroma,[20] branchial maldevelopmental anomaly,[21] mixed tumors,[22, 23] reticulosarcoma,[24] lymphosarcoma and squamous cell epithelioma.[25, 26]

The primary symptom in this case was cough, a constant sign of vagal irritation.[27]

In five reported cases of removal of neuromas of various types from the lateral pharyngeal wall, Horner's syndrome[17, 28] subsequently occurred. In three cases the syndrome was merely transient,[16, 20, 29] indicating involvement of the adjacent cervical sympathetic chain. In three of these cases there was an ipsilateral abductor vocal-cord paralysis;[16, 17, 29] in Turchik's case it had been present for five years prior to operation.

It is therefore postulated that the "warning signal"—in this case, cough—was caused by vagal irritation transmitted by way of visceral sensory fibers to the pharynx from its nodose ganglion. The increased symptoms at night might be attributed to engorgement of the tumor when the patient was recumbent, and the decrease in symptoms might be ascribed to the effect of upright posture on the tumor.

Neither Horner's syndrome nor paralysis of the vocal cords occurred postoperatively in the case herein reported.

Although hemangiomas are classified as benign tumors, it has been repeatedly pointed out that they should be surgically treated, if only to prove the nonmalignancy of the particular lesion. They are likely to grow in size, with increasing local discomfort in the pharynx, increasing danger of hemorrhage and the possibility of vocal-cord paralysis. Figi recently has reviewed modes of therapy;[30] in the case reported herein excision and diathermy plus irradiation were used.

SUMMARY

A case of hemangioma of the lateral pharyngeal band is reported. The case is considered of interest because of the relative rarity of such pharyngeal tumors, the circumscribed location of the lesion and its unusually inconspicuous appearance clinically; these points have been discussed. The pertinent literature has been re-

viewed briefly. The cause of the patient's chief complaint, cough, was hypothesized to be vagal irritation. The reasons for adequate surgical treatment have been listed.

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L

FIBROSARCOMA WITH UNDERLYING LEUKOPLAKIA OF THE LARYNX FOLLOWED TWENTY-TWO MONTHS LATER BY SQUAMOUS CELL CARCINOMA

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The occurrence of a fibrosarcoma and leukoplakia followed 22 months later by an epidermoid carcinoma within so small an organ as the larynx is of interest.

Mrs. M. O'T, aged 72, was first seen on June 6, 1944. She had urgent dyspnea which had been developing for the last eight weeks. Prior to this she had been hoarse for an indefinite number of years. She was a delightful but garrulous Irish lady who woke talking and continued to talk each day until sleep overcame her.

Mirror examination of her larynx showed a smooth, pinkish-gray mass somewhat the size and shape of a jelly bean, filling the larynx and obscuring the vocal cords and ventricular bands. There was no adenopathy in her neck and the physical examination was otherwise negative. As her dyspnea was urgent, a direct laryngoscopy was done and the mass was grasped with large cup forceps and removed. Immediately she breathed well. The site of the attachment could not be determined. There was, however, a leukoplakia extending from the anterior commissure almost the entire length of the left cord and over the anterior half of the right cord. The tumor mass was smooth, pinkish-gray and had the feeling of a fibroma.

The pathologist's report follows: "The specimen consisted of a nodular structure, the outer surface white and smooth, measuring 2 x 1.2 x 1 cm. The cut surface is smooth, homogeneous, translucent, and of a grayish-white color. The microscopic examination showed tissue which is moderately cellular and composed of spindle-shaped cells arranged in irregular interlacing bundles. The stroma is loose, edematous and pale, pinkish in color. The cells show considerable variation. There are moderate numbers of giant cells, and

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Presented at the New England Otological and Laryngological Society, March 31, 1948.

mitotic figures are common. The structure is covered in part by surface epithelium, which is keratinizing, and consists of tumor extending up to the surface showing no evidence of encapsulation. Diagnosis: Fibrosarcoma."

For the next four months her larynx was carefully watched. The area of leukoplakia seemed to be gradually extending. On November 11, 1944, a second direct laryngoscopy was done, and the pathologists considered the tissue removed to be a hyperkeratosis or leukoplakia.

The pathologist's report follows: "The specimen consists of small, whitish piece of tissue, measuring 5 x 1 x 1 cm. The microscopic examination showed epithelium with little submucosal tissue present where there is a narrow rim in which there are a few polynuclear leukocytes. The epithelium, however, is quite markedly thickened. There is some edema of the epithelium in the squamous portion and in the basal layers, while in general it is covered by a very thick keratin. There is no mitotic activity and there are no areas of invasion. Diagnosis: Hyperkeratosis (Leukoplakia)."

Her larynx then was observed at intervals for the next 15 months. The area of leukoplakia was increasing in the anterior commissure and on the ventricular bands. In the last month an outgrowth was noted extending from the left ventricular band. On April 2, 1946, a third direct laryngoscopy was performed and the pathologists considered the tissue removed to be a squamous cell carcinoma, grade one.

The pathologist's report follows: "The specimen consists of two whitish pieces of tissue which have been fixed in formalin, measuring 0.5 cm. in diameter and relatively friable. The microscopic examination shows that in these sections there is a tumor which differs considerably in morphology from the original tissue reported as fibrosarcoma. This consists of irregular groups of squamous cells which are frequently forming epithelial pearls; mitotic figures are occasionally present and the tumor cells are infiltrating the underlying tissue. The surface epithelium shows some thickening and hyperkeratosis. Diagnosis: Squamous Cell Carcinoma, Grade One."

On April 8, 1946, 22 months after the removal of the fibrosarcoma from her larynx, a laryngectomy was performed, as it was felt that this was the only procedure that would remove the total area of hyperkeratosis in which the carcinoma developed, and that x-ray treatment would be ineffectual. The pathologists considered the growth to be a squamous cell carcinoma, grade two.

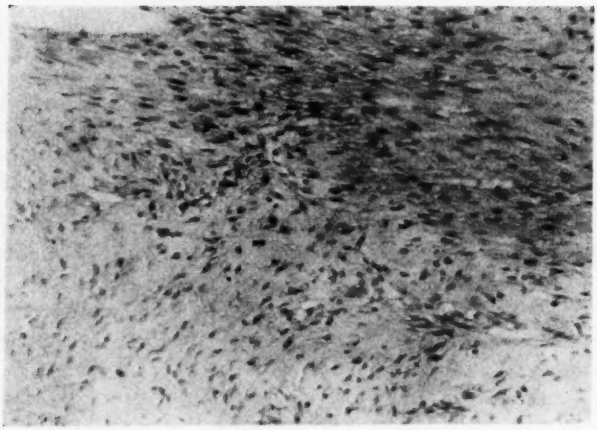

Fig. 1.—Photomicrograph of section from pedunculated mass removed at direct laryngoscopy. The appearance is characteristic of fibrosarcoma.

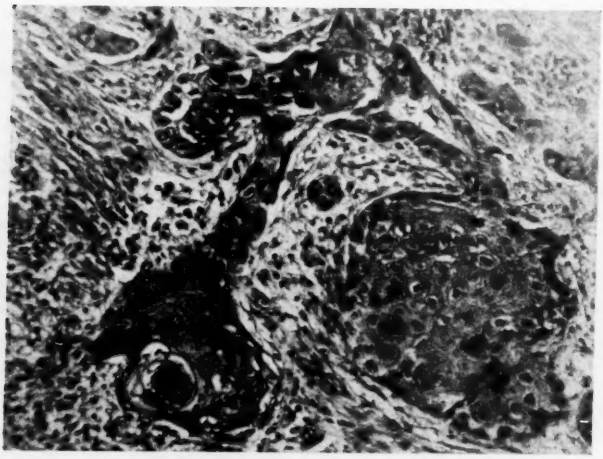

Fig. 2.—Photomicrograph of section from tissue removed at direct laryngoscopy twenty-two months later. The appearance is characteristic of a Grade 1 squamous-cell carcinoma.

The pathologist's report follows: "The specimen consists of a larynx with epiglottis attached. There is an ulcerative lesion on the left vocal cord which extends from the anterior commissure back along the border of the cord for 1 cm. and up onto the left ventricular band for 1 cm. On the right vocal cord leukoplakia extends from the anterior commissure back along the vocal cord for a distance of 12 mm. and up onto the ventricular band for about 8 mm. These lesions join in the midline. The microscopic examination showed a tumor composed of irregular masses of squamous cells, tending to grow down into the surface epithelium. The mucosa in part is ulcerated and covered with exudate. The tumor extends deeply into the wall and almost reaches the outermost lining. There is a rather heavy inflammatory infiltrate throughout the tissue, groups of tumor cells are occasionally seen forming epithelial pearls, and occasional mitotic figures are seen. Diagnosis: Squamous Cell Carcinoma, Grade Two."

The patient has made an uneventful recovery and is developing an esophageal voice. It is probable that with her ability as a conversationalist and the history of hoarseness of years' duration that the original lesion was a fibrohemangioma which later underwent sarcomatous changes. Low-grade fibrosarcomas increase in size slowly and it is probable that the constant irritation of the pedunculated tumor mass brought on changes in the epithelium of the vocal cord and ventricular bands that resulted in an extensive leukoplakia. This was a precancerous lesion.

Since 1940, 25 cases of sarcoma of the larynx have been reported in the literature. Sixteen of these were fibrosarcoma. It is generally agreed that these are most frequently slow growing, pedunculated growths with little tendency to metastasize, that the treatment of such pedunculated growths is removal with electrocoagulation of the base and that observation of the patient at frequent intervals, thereafter, is necessary.

The history of this case suggests that the frequently seen fibrohemangioma arising at the junction of the anterior third and the posterior two thirds of a vocal cord may on occasion be a prefibrosarcomatous lesion.

Clerf in an excellent article points out that keratosis of the larynx, one type of which is frequently called leukoplakia, is a precancerous lesion, as it proved to be in this case.

47 DEERING STREET.

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LI

EOSINOPHILIC GRANULOMA OF TEMPORAL BONE ASSOCIATED WITH DIABETES INSIPIDUS

A CASE REPORT

J. Lewis Dill, M.D.

Detroit, Mich.

The bony lesion, eosinophilic granuloma, was first described as a clinical entity in 1940, though similar lesions had been reported earlier. In that year the reports of Otani and Ehrlich[1] and Lichtenstein and Jaffe[2] appeared almost simultaneously.

Since then, a number of cases, usually single, have appeared in the literature. It is interesting to note the frequency with which cases have occurred in the armed forces. Dundon, Williams and Laipply[3] report five and state that up to July 1, 1945, there were 48 cases reported in the literature. In the same journal there is a report by Hamilton, Barner, Kennedy and McCort[4] of nine cases seen in the army hospitals. Two cases are reported by Michael and Norcross,[5] one case by Weaver and Carter[6] and one case in a child of 12 weeks of age by Kendrick and Woodruff.[7]

The cause of the lesion is not known, but is considered to be most probably of an inflammatory nature. There does not appear to be a familial tendency. Practically every bone in the body has been found to harbor the granuloma, with the exception of the carpals, metacarpals and phalanges, and the corresponding bones of the feet.

The most common symptom is mild local pain, associated with swelling of the soft tissues, and tenderness. Among the systemic manifestations there may be slight fever, anorexia, headache, loss of weight and moderate leucocytosis. In a limited number of cases there was found an increase in eosinophils of the blood and two cases have been associated with diabetes insipidus.

The main histological components of the granuloma are stated by Otani and Ehrlich[1] as the following:

1. Large pale cells, usually in reticular arrangement.

Fig. 1.—Roentgenogram of the mastoids made June 25, 1945. Note area of destruction (outlined by arrows) of the right temporal bone, superior to the mastoid.

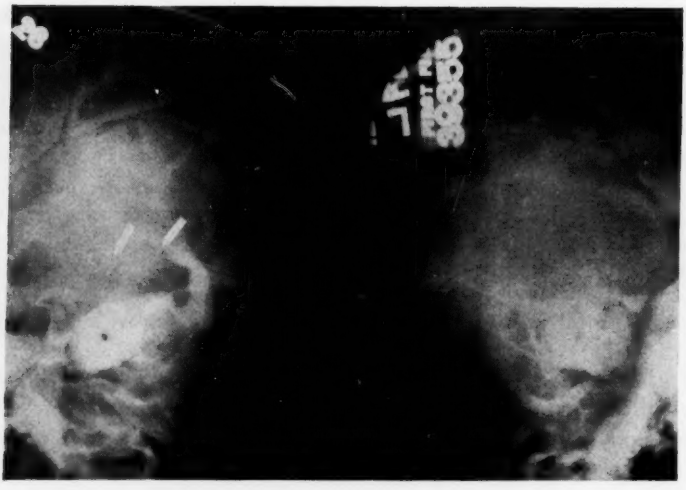

Fig. 2.—Roentgenogram of the mastoids made December 30, 1946. Note increased area of destruction (outlined by arrows) of the right temporal bone.

2. Leucocytes, mainly neutrophiles, but also many lymphocytes and eosinophils.
3. Multinucleated giant cells, mainly of the osteoclastic type.
4. Products of destruction: necrotic débris, broken-down fragments of bone, blood and blood pigment, lipophages.
5. Products of repair: fibrous tissue and callous bone.

Three stages of this disease have been considered by some authors as: (a) the early destructive or cystic stage; (b) the intermediary stage in which the nuclear cells have vacuolated (foamy) cytoplasm and eosinophils are few in number or absent; (c) the late stage with proliferation of connective tissue and eventually regeneration of bone. The mononuclear histiocyte or foam cell is considered the characteristic cell and causes speculation on the relationship between eosinophilic granuloma, Letterer-Siwe's and Hand-Schüller-Christian's disease.

The diagnosis can be made only after an operation and pathological diagnosis of the tissues. Treatment consists of surgery, irradiation, or a combination of both. A few cases are reported in which the patient has recovered spontaneously, after surgery or roentgen therapy. Surgery is advisable in order to make an accurate diagnosis.

REPORT OF A CASE

B. Y., (No. 393557), a white male, 2½ years of age, was first seen in the Department of Pediatrics in July 1943, at which time the diagnosis was diabetes insipidus. There was no note of any ear infection and no history of otitis media was obtained. The hemoglobin was 12.1 gm., red blood count 4,390,000, white blood count 12,360, polymorphonuclears 37, small lymphocytes 47, large lymphocytes 12, eosinophils 3, mononuclears 1.

On June 23, 1945, he was seen in the Ear, Nose and Throat Clinic with the history of a draining right ear of 14 months' duration. Examination of the ear showed a purulent discharge in the canal and a swelling of the posterior canal wall, resembling a furuncle; the ear drum was not visualized.

A roentgenogram of the right mastoid showed clouding of the mastoid with evidence of some destruction (Fig. 1). A mastoidectomy was advised but the parents refused this.

On March 5, 1946, the tonsils and adenoids were removed. At that time the right ear was still draining. There was what appeared

Fig. 3.—Roentgenogram of the skull made February 20, 1947. Note oval area of destruction of the right parietal bone.

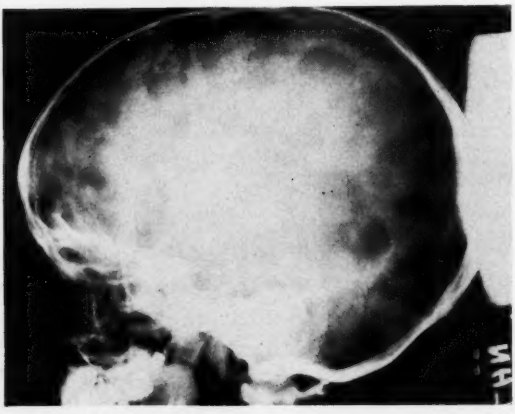

Fig. 4.—Roentgenogram of the skull made September 30, 1947. The areas of destruction in the right parietal and temporal bones appear to be calcified.

to be a furuncle of the posterior auditory canal wall. On December 30, 1946, the furuncle of this region was again noted and diagnosed as a fistula, with whitish material resembling a cholesteatoma seen in the orifice of the fistula. There was slight discharge in the canal. The right ear drum was intact and scarred. No swelling was present but slight tenderness was elicited over the right mastoid area.

Roentgenograms showed sclerosis throughout the extent of the right mastoid. No cells were visualized. There was a large irregular area of destruction of the temporal bone above the mastoid (Fig. 2).

On January 24, 1947, the patient was admitted to the hospital. The general physical examination was negative. The hemoglobin was 11.0 gm., red blood count 3,160,000, white blood count 18,500, polymorphonuclears 60, lymphocytes 38, eosinophils 2.

A right simple mastoidectomy was performed. The mastoid cortex was intact and sclerotic. A large, rather oval, somewhat irregular area of the temporal bone was found destroyed and the dura exposed. This exposed dura comprised an area extending from an area posterior to the sinodural angle, anteriorly beyond the temporomandibular joint and superiorly about one inch above the mastoid (Fig. 2.) The dura was markedly thickened, and in some areas dirty granulations were present. The mastoid cells had been completely destroyed and replaced by a whitish cholesteatomatous material. This cholesteatoma was protruding through the fistula of the posterior canal wall. In a few areas dirty granulations were noted and removed. These granulations failed to bleed on removal.

The child was given a transfusion of whole blood. Penicillin, 25,000 units, was given intramuscularly every three hours for four days. Recovery was uneventful except for an attack of chickenpox.

Pathological Report.—"Diagnosis: Eosinophilic Granuloma. There are fragments of cancellous bone about which are areas of dense fibrous tissue and loosely reticular fibrous tissue infiltrated with lymphocytes, larger, oval mononuclear cells with rounded or indented, uniform nuclei and large polyhedron-shaped cells having pale foamy cytoplasm and small, dark, central nucleus. Some bits of tissue submitted consist almost entirely of these foamy appearing lipophages or xanthoma cells. The cells are separated by a delicate reticular network which in some areas is more fibrous in character. In addition there are bits of tissue consisting almost entirely of eosinophils which in some areas are disposed in compact sheets and in other areas more loosely arranged and connected by thin, delicate, cytoplasmic bridges. Within these masses of eosinophilic cells are

circumscribed areas of necrosis. In some instances the morphologic detail is completely destroyed, while in other areas circumscribed pink dots, apparently necrotic eosinophils, can be seen. Scattered through these eosinophilic masses are large mononuclear cells with oval or indented nuclei, vesicular nuclear in character, and having variable amounts of pink-staining cytoplasm. In some instances cells of this type are fused to form small, multinucleated, giant cells containing two to six nuclei. The histologic detail of this lesion is in all respects comparable to that described in the literature as eosinophilic granuloma. Recent reports, however, indicate a close relationship between this lesion and other xanthomatoses of bone—more specifically Hand-Schüller-Christian disease."

On February 17, 1947, the mastoid incision was well healed. The fistula of the right auditory canal was healed, the drum was intact, and the patient heard the whispered voice. On February 20, 1947, a small, oval, raised, tender area was noted in the right parietal region 1½ inches posterior to the hair line. The roentgenogram revealed a rounded shadow of decalcification, about 1.5 cm. in diameter, in the right anterior parietal region. There was also evidence of similar decalcification in the base of the right parietal and occipital bones just above the mastoid (Fig. 3). Roentgenograms of the chest, pelvis, spine and long bones of the extremities were negative for areas of decalcification.

X-ray therapy to the areas of decalcification was given as follows: From February 25 to March 11 a total of 400 r in air were given to each of two areas of the skull (right mastoid area, 6 x 8 cm., and right parietal area, 3 cm. circle). The dosage was administered in 100 r fractions at 3 to 4 day intervals. Technical factors were 140 K.V., 6 ma; ¼ mm. cu. and 1 mm. Al filtration; H.V.L. 0.5 cu. r per minute—39. F.S.D.—24 cm.

On September 30, 1947, the roentgenograms showed the round defect in the right parietal area to be very indistinct. The area above the right mastoid was also very indistinct. These areas appear to have recalcified (Fig. 4).

The child has remained well up to the present, but no effect on the diabetes insipidus has been noted. The boy is rather small for his age but is bright and rather precocious.

SUMMARY

A case of eosinophilic granuloma is reported in a boy of five years of age, who had diabetes insipidus since 2½ years of age. The

onset of the mastoid involvement is not definitely known, though this boy had a fistula of the auditory canal for many months. A secondary lesion in the right parietal bone was noted following operation. The white blood count was elevated but eosinophils were not increased. The blood cholesterol was normal. Both lesions appear to have healed following surgery and roentgen therapy.

HENRY FORD HOSPITAL.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, December 1, 1947

THE PRESIDENT, DR. JOHN R. LINDSAY, IN THE CHAIR

Benign Cysts of the Paranasal Sinuses

HAROLD F. SCHUKNECHT, M.D.

CHICAGO, ILL.

Benign cysts of the paranasal sinuses are of two general types, those which take origin from the sinus mucosa and those which invade the maxillary sinuses from the teeth. From the mucosa arise three types: nonsecreting cysts, mucous gland (retention) cysts and mucoceles. Nonsecreting cysts are found in the maxillary sinuses and occur in about five per cent as shown by routine x-ray studies. On x-ray films they produce a characteristic dome-shaped shadow, but may also fill the sinus and produce complete homogeneous clouding. The cyst forms as a collection of straw-colored fluid in the loose subepithelial connective tissue of the mucosa. This fluid is usually sterile and coagulates on standing. Small nonsecreting cysts usually produce no symptoms and need not be removed. When the cyst occupies one half or more of the sinus, there may result not only local symptoms but general symptoms of fatigue, irritability and headache. Having no epithelial lining, this cyst may disappear after spontaneous rupture, or, puncture and lavage. Removal of part or all of the cyst wall by an antrum window operation usually results in a cure. Occasionally radical removal of the sinus contents is necessary.

The mucous gland (retention) cysts occur in all the sinuses. They are small, not individually evident on x-ray study, have an epithelial lining, and appear as part of the histopathologic picture in chronic hyperplastic sinusitis. Hyperplasia of the epithelium of the duct of the acinotubular gland is probably an important factor

in producing stenosis with resulting retention of secretion and cyst formation. At the time of operation these small cysts are often misinterpreted as being small abscesses.

Mucoceles occur commonly in the frontal and ethmoid sinuses. Obstruction of the nasofrontal duct or an ethmoid ostium probably occurs in all cases of true mucocele. Repeated acute infections, in some instances accompanied by temporary blockage, appear to increase the probability of eventual permanent blockage and mucocele formation. As the products of secretion, inflammation and desquamation accumulate in the cystic space, it expands and encroaches upon the nasal cavity, orbit and anterior cranial fossa. The cyst invades the orbit early by pressure erosion of the floor of the sinus with characteristic proptosis and lateral and downward displacement of the eye. Acute infection of a mucocele (pyocele) may be a serious complication if there has been expansion of the cyst into the anterior cranial fossa. Chronic mild infection of a mucocele may result in a chronic hyperplastic osteomyelitis, in which case the cystic space may be almost filled with new bone. An intranasal operation may be adequate; however, usually a radical external fronto-ethmoidal sinusotomy is necessary. An opening into the nose which will remain open must be created.

Odontogenic cysts are of two types: the follicular cyst and the radicular (root) cyst. The follicular cyst arises from the enamel organ epithelium of the dental follicle. When one or more teeth are present, it is commonly termed a dentigerous cyst. The cyst will not contain a tooth if it develops from the enamel epithelium before it has become sufficiently differentiated to form enamel. The lining epithelium is stratified squamous in type and closely resembles oral epithelium. As the cyst expands, it may invade and fill the maxillary sinus. If a tooth is present in its wall, it may be displaced into the sinus. The radicular cyst develops from the apical or root granuloma. Epithelial cell rests of the periodontal membrane may be included in the root granuloma and by inflammatory stimulation may completely line the central cystic space as it forms. Radical removal of the sinus contents is necessary for both types of odontogenic cysts; for this the Caldwell-Luc operation is generally best suited.

DISCUSSION

DR. WALTER THEOBALD: I wonder if many of these cysts do not get well by simple lavage. I have found that, after a few irrigations with return of straw-colored fluid, the cyst wall may wash

out of the opening itself or into the middle meatus so that it could be removed. I think furthermore that the window operation does suffice in the majority of cases for removal of these cysts.

DR. SAMUEL SALINGER: What is the significance of the simple inclusion cyst in so far as the patient is concerned? It has been my experience that in most cases they cause little trouble and are often discovered accidentally on routine x-ray examination. Frequently they disappear spontaneously and occasionally are punctured during an antrum irrigation. In my opinion they are not very significant unless they attain sufficient size or block the ostium. The case of the mucocele is entirely different; these should be removed. The question as to what procedure is best is a matter for another discussion.

DR. JOHN R. LINDSAY: These nonsecreting cysts have interested me for some time, probably because we have encountered a few cases where they were not so benign as had been thought. Cultures of the straw-colored fluid were usually sterile. We studied the chemistry of these fluids a few years ago; they have a high protein content, corresponding to exudates rather than transudates. When they are small, perhaps less than half filling the sinus, they are probably innocent. Yet I have had the experience of puncturing one cyst with a lumbar puncture needle and withdrawing about 10 cc. of fluid, and the next day the patient was in bed with acute arthritis. I have had other experiences where simply letting the fluid out relieved acute pain in the region of a joint. We have had other cases in which there was no evidence that the cyst was producing symptoms. Some will disappear after irrigation. They do not have an epithelial lining and may collapse and disappear. Others recur and require an antrum window operation to permit removal; still others require radical antrotomy for adequate removal. I have observed cases in which a window operation had to be followed by radical exenteration of the sinus mucosa to prevent recurrences and relieve the accompanying distress. When there is evidence of much hyperplastic change of the mucosa in addition to the cyst, it is preferable to do the radical operation than to risk having to do a second operative procedure.

DR. MAURICE F. SNITMAN: Does Dr. Schuknecht include cystic adenomata among the benign cysts of the maxillary sinus?

DR. SHERMAN SHAPIRO: Do these benign cysts ever undergo malignant degeneration?

DR. HAROLD F. SCHUKNECHT (closing): Dermoids do very frequently involve the nasal structures, but rarely the paranasal

sinuses proper. I think the only type that becomes malignant is the follicular cyst. Kahn, in 1933, reported several cases in which follicular cysts apparently developed into adamantinomas.

Fundametal Conceptual Changes in Regard to Chronic Suppurative Sinusitis

HENRY L. WILLIAMS, M.D.

ROCHESTER, MINN.

(Abstract)

In the last 25 years more fundamental changes have taken place in the organized body of concepts that form the philosophy of chronic suppurative disease of the paranasal sinuses than in any other division of otorhinology. These conceptual changes fall into two groups. The first is that having to do with the clinical pattern of chronic suppurative disease of the paranasal sinuses. Formerly, chronic suppurative sinusitis was thought to be characterized by severe pain in the head or headache, and long papers were devoted to the description of the headache pathognomonic of suppuration in certain particular sinuses; it was considered to be due to pressure of retained secretions on the wall of the sinus.

As a result of investigative work by Thomas Lewis, Kellgren, H. G. Wolff and his co-workers, and Behnke, it is now generally recognized that as a rule chronic suppurative sinusitis is a painless disease. Exceptions that "prove the rule" do occur, and it is important to recognize them clinically.

When chronic suppurative sinusitis coexists with clinical evidence of chronic vasomotor rhinitis, however, the condition is quite different. This combination of disorders is termed by the present author "chronic hyperplastic sinusitis." The clinical pattern is characterized by the prominence of the symptom of headache. Because patients who have allergic involvement of the paranasal sinuses without symptoms and signs of suppuration are seen, in whom the symptom of headache is a prominent clinical feature, and because pain in the head (or headache) is an unusual clinical feature in patients with an uncomplicated chronic sinusitis, it would seem reasonable to conclude that headache in chronic hyperplastic sinusitis results from the allergy rather than from the suppuration. By clinical examination it can then be ascertained that the headache does

not arise from the sinuses, but usually from other pain-sensitive structures about the head, as a rule the cranial and cervical musculature and the extracranial blood vessels. It would appear, then, that to secure clinical relief equal attention must be given to both the allergic and the suppurative factors.

The so-called routine allergic management in hyperplastic sinusitis has proved disappointing. Positive results of skin tests rarely give specific information, generally indicating only that the patient under consideration is pathergic; attempts at relief by specific desensitization usually fail. In chronic hyperplastic sinusitis, diagnosis by means of elimination diets also has proved ineffective. The author believes these failures to result from the fact that in this syndrome what is presented generally is not an antigen-antibody type of reaction but, rather, so-called physical allergy for which the term "pathergy" has been suggested by Urbach. This disturbance appears to be failure of the general adaptation reaction described by Selye, the normal physiologic process proceeding no further than the stage of "alarm." Because of cellular injury, histamine is released in the alarm reaction, producing a clinical picture indistinguishable from that of "allergy."

It is imperative that the allergic treatment of a patient with such sinusitis be carried out by, or in close association with, the rhinologist. So-called nonspecific allergic treatment is usually more successful in relieving the allergic symptoms than so-called specific methods. Among nonspecific methods are included subcutaneous injection of foreign proteins such as typhoid and milk and the use of house dust. The low dosage method of Hansel is favored. Symptomatic relief has been obtained for a number of patients by the use of capillary vasodilators such as monoethanole-amine nicotinate (nicamin), sodium nicotinate, nicotinic acid, histamine, neostigmine, magnesium sulfate and the like. Nicamin is preferred because of ease of administration.

Certain rhinologists maintain that there is an allergic factor present in nearly all cases of chronic suppurative sinusitis. At the Mayo Clinic, however, microscopic examination of sections of mucosal lining and other tissue removed at operation in cases diagnosed as chronic suppurative sinusitis, almost invariably revealed an inflammatory picture, thus confirming the so-called allergic picture. The role of the eosinophil in allergy is unknown, and it is questionable if its presence in examined tissue is a sufficient basis for an unqualified diagnosis of allergy.

Formerly, it was felt that free and adequate drainage of the rigid-walled cavities was the prime consideration, and to this end the functioning structures of the nasal chambers, the mucosa and the turbinal tissues were freely sacrificed. Thirty years ago Lillie was one of the first to protest against this indiscriminate surgery. Physiologic investigations under his guidance were undertaken by Hansel, Stark, Heetderks, Hilding and others. Proetz, Van Alyea and others have added greatly to the knowledge of nasal physiology and its importance to the well-being of the individual. It is now generally accepted that while adequate drainage must be secured, even more important is preservation of the functioning nasal tissues. It is now generally recognized that the operative procedure should be adequate enough to achieve drainage and aeration, and at the same time rational enough to preserve the functioning structures of the nose. In the condition defined as chronic hyperplastic sinusitis a judicious combination of surgical and allergic treatment must be used to give symptomatic relief.

Several technical changes in operations on the various sinuses have resulted from this changing philosophy. There is a trend toward the naso-antral window in operations on the maxillary sinus; in most instances removing the lining mucosa is contraindicated. The ethmoid sinuses are almost invariably involved along with other sinuses; in simultaneous involvement of the maxillary sinus and the anterior ethmoid sinus, a naso-antral window frequently is sufficient to relieve both disturbances. In suppuration of the frontal and ethmoid sinuses, however, exenteration of the ethmoid sinuses is necessary to afford access to the frontal sinus. Chronic fronto-ethmoid suppuration, in the author's opinion, does not lend itself to preservation of the mucosa of the frontonasal duct, as proposed by Van Alyea. In most instances reactive osteitis tends to produce progressive constriction of the frontonasal duct, occasionally resulting in formation of a pyocele. As a rule, therefore, intranasal fronto-ethmoid exenteration is the procedure of choice, though external fronto-ethmoid exenteration requires less skill and experience and is potentially less dangerous. Another factor in favor of the intranasal procedure is the greatly diminished tendency for scar tissue to close the nasofrontal opening, one of the principal causes of failure in external operations on the frontal sinus. This is because of wide destruction of the frontal process of the nasal bone and removal of the lamina papyracea of the ethmoid, as usually practiced in the Lynch technique. The first procedure allows collapse of soft tissue into the approach to the frontonasal opening, while removal of the lamina papyracea favors formation of excessive granulation tissue

from the periorbita, with subsequent closure of the frontonasal opening by scar tissue.

External operation on the frontal sinus is justified when the disease has perforated one of the walls of this sinus with formation of an abscess or fistula, when there is evidence of partitioning in the frontal sinus by bony laminae, or in cases in which reactive osteitis has so hardened the bone in the area that dangerous force would be necessary in the performance of an intranasal procedure.

Continuance of the external incision around the inner canthus of the eye favors involvement of the venous plexus around the lacrimal sac, the importance of which in promoting spreading osteomyelitis of the frontal bone and osteitis of the facial bones has been pointed out by Lillie. With the use of tantalum foil, shaped to fit the frontonasal opening at the time of operation, the author has found that closure of this aperture can be avoided.

DISCUSSION

Dr. O. E. Van Alyea: I agree heartily with almost everything Dr. Williams has said. All who do much work in rhinology and laryngology are becoming "allergy-minded." As is often the case, however, when a new idea takes hold there is a rush to put it into practice and it becomes a fad with many people who overlook the wide-range management of the cases. The reports from dust desensitization have been amazing. Yet are we not all somewhat inclined to regard every case as allergic, and overlook some other fundamental factors in the disease and in the symptom complex?

In surgical procedures on the frontal sinus, Dr. Williams displays a leaning toward intranasal methods when possible. Of course it has always been consistent to include with procedures on the frontal, exenteration of the anterior ethmoid sinuses. I have never been able to understand why the whole group of ethmoid cells need be sacrificed. It is true that certain cells en route to the frontal sinus, frontal cells or ethmofrontal cells, near the ostium, are of major importance. In my opinion we should give up the idea of removing bullar cells; they never block the frontal sinus, and we disturb an area which may be free of infection. However, cells which do block drainage must be removed, by passage of a cannula or other means. Those in the lumen of the sinuses may be taken care of through the external frontal approach without disturbing the sinus or the ostium.

During the past few years we have studied some old sections removed by radical surgery and found, amazingly enough, that these contained a preponderance of eosinophils. The results of surgery in these cases were temporary; many of these patients are now chronic nasal wrecks. Those of them that we still see occasionally in the dispensary are trying to get relief from symptoms that are worse now than before radical surgery.

Penicillin in my hands has been a failure by the Proetz method and I now reserve it for cases in which there are serious symptoms.

Dr. Williams did not mention the "irreversible" changes referred to constantly in the literature. I have not encountered any. My experience has been similar to his; when the antrum is opened by way of a window procedure and thickened membrane is found, recovery takes place when drainage is established. Is this an irreversible change? We hear frequently of little abscesses between the sinus and the bone. In our examination of specimens we failed to find any abscesses in extirpated mucosa, nor did we find ulcers or other conditions that have been described.

Another item Dr. Williams failed to mention is the so-called ethmoiditis. We see conditions of multiple polyposis of the middle meatus; yet on examination of specimens from the anatomic laboratory, polyps in the middle meatus usually arise from the maxillary ostium, the uncinate process and the bulla. I have yet to find one in which polyps could be traced to the ethmoid labyrinth.

DR. SHERMAN SHAPIRO: I should like to ask Dr. Williams if he considers that complete obliteration of the frontal sinus can be achieved by any method short of removal of the entire anterior wall and anterior floor, including the septum. I should also like to ask if there is any postmortem material at the Foundation or elsewhere showing that obliterated frontal sinuses have remained so.

DR. SAMUEL SALINGER: To the older men in this audience this subject is of extreme interest because we have seen a remarkable evolution in the general concept of sinus disease; the younger men know only half the story. The other half is buried in textbooks, voluminous literature and the memory of some of us. I well recall when discussions were confined solely to surgical procedures with arguments as to which was best. The volume of sinus surgery was tremendous. One member of this Society reported 300 intranasal frontal sinus operations performed with the rasp. My own predecessor, Dr. Otto Freer, was in favor of a motor-driven burr with special curettes of his own. The Halle operation was widely em-

ployed, often with disastrous results. Then came the external fronto-ethmoid exenteration which was extensively employed, and of which we hear very little of late.

The trend to conservatism has been definite and unmistakable. Year by year in our own practice we do fewer and fewer operations and so-called radical sinus surgery is becoming almost as rare as the simple mastoidectomy. Increasing knowledge of the physiology of the nose and of the constitutional factors giving rise to nasal symptoms have explained many of the bad results which formerly followed surgery.

I agree with Dr. Williams that allergy studies have often proven disappointing except in certain types of cases. We have found them more significant in children and adolescents than in adults, probably because of nutritional and endocrine factors.

There is one item I would like to stress; the time to begin treatment of a chronic sinusitis is when the acute attack subsides. We are prone to dismiss our patients too soon. It is important at this time to check the sinuses thoroughly, ascertain their functional capacity and examine the patient for factors which might predispose him to future attacks with increasing damage to the lining mucosa.

I should like Dr. Williams, if he will, to state more explicitly just what he considers the proper indications for intranasal antrostomy.

DR. MAURICE F. SNITMAN: In a study of more than 200 specimens of nasal mucosa removed during radical sinus surgery, we have found typical chronic inflammation present in only two instances. In most cases the microscopic picture was that of edema associated with varying degrees of eosinophilia.

DR. G. H. MUNDT: It seems to me there are some indications for going into the maxillary sinus through the canine fossa. It is a simple procedure and one gets a much better view than by other means. There is little reaction, general or local, and one can make a satisfactory opening into the inferior meatus without destruction of the inferior turbinate. I have long since discontinued trying to remove all the mucosa from the maxillary sinus.

I should like to ask Dr. Williams to speak on antihistamine drugs.

DR. ROBERT H. FRASER: In diagnosing conditions of the maxillary sinus the number of positive eosinophil smears is very revealing; next comes x-ray study and, if needed, diagnostic irrigation. The more severe infections, even some of dental origin, seem, in my hands, to yield to irrigations with the instillation of 2 cc. of penicillin in saline (using 10,000 units per cc.) ordinarily at six-day intervals until negative. The empyema may be at an end and in time its concomitant derangements, with the result that allergy proceeds further. At this point iodized oil reveals pedunculations and the surgeon may abandon the window for the canine fossa approach. I believe the necessity of surgery must be faced, although the number of cases operated upon will be only a fraction of those which would have been operated upon in the twenties.

With Dr. Williams we must all recognize chronic frontal sinusitis which necessitates external operation with, perhaps, employment of the Riedl collapse type of obliteration. I should like to recall the motion picture presentation of Simpson before the Academy in 1943. He has had a large and remarkably successful series through employment of the Lynch approach, which his presentation showed to lead to prompt and permanent healing, including obliteration of the cavity on the blood clot principle. I can vouch for the preoperative data and the satisfactory healing of a good number of his cases. Integral to the success are a number of Simpson requirements. The elimination of mucoperiosteal remnants both in the frontal sinus and the ethmoid is precisely and meticulously done.

The operator works within the sinus by mirror vision to achieve finished surfaces. The newer detail includes a motor-driven burr for irregularities. The nasofrontal duct is entirely removed. The middle turbinate is left intact. Very little packing is used and then only in the nasal fossa for 24 to 48 hours, after which no treatment whatsoever is carried out except slight cleansing of the nose itself. The frontal sinus cavity is not disturbed following operation. With all the floor of the frontal sinus removed and the ethmoid exenterated, the inferior aspect of the operative field can be disregarded as there can be neither expansion nor constriction of any outlet and granulations need not be feared. It is not expected that the sinus area will be re-entered at any time. Penicillin, since it has become available, is administered for seven or eight days, furthering the successful organization of the blood clot in order to obtain obliteration of the cavity. I know of one case of an operation being done by this method without incident to an anteroposterior depth of 18 mm. I am sure Dr. Simpson has made valuable contributions which are capable of formulation and practical application.

DR. HENRY L. WILLIAMS (closing): I want to thank all of you for the full discussion. I do not know that a diagnosis of allergy is justifiable in every instance in which a large number of eosinophils are found in the mucosa on histologic examination. The role of the eosinophil in allergy is not known. Code and others have suggested that the eosinophil is the principal source of histamine, but I do not believe the finding of many eosinophils in the nasal mucosa on microscopic examination unqualifiedly indicates that the individual has allergic disease; in an occasional case there could be reasonable doubt. The histologic or cytologic diagnosis of allergy cannot always be substantiated by a trial of routine allergic methods of treatment, or antihistamine drugs or other nonspecific anti-allergic treatment. It seems to me, therefore, that the mere presence of eosinophils in nasal smears or histologic sections is insufficient data on which to base a diagnosis of allergy.

The same statement can be made about the presence of polypoid formation. Polyposis results when fluids escape from the blood vessels into the pericellular tissues and edema results. Where underlying tissue is deficient in fibrous and elastic tissue, the edematous part is not supported, hangs down and forms a polyp, not hyperplasia. As Dr. Galloway has said, "in the occasional case when sufficient aeration and drainage are given" evidence of allergy disappears and the appearance of the nasal mucosa returns to normal. If one were an allergist, one might argue that bacterial allergy produced the polyps which disappeared on eradication of the infection. It is difficult to prove that hypothesis one way or the other.

It can be shown that allergic symptoms do not always demonstrate that a child is allergic. Children on an abnormal diet sometimes have symptoms of vasomotor rhinitis which are not on an allergic basis. Dr. Arild Hansen indicated the role of lack of unsaturated fatty acids in producing these symptoms. The rhinitis sometimes clears up on administration of unsaturated fatty acids. If children eat fat meat, they will not have this type of nasal congestion, but if they will not eat it, a teaspoonful of lard mixed with sugar and spice given as candy three times a day will relieve the symptoms and give them their quota of fatty acids. Butter has a very low iodine number and cannot be substituted. Corn oil is good. Edema of the nasal mucosa may be associated with low basal metabolic rates among both adults and children, and oftentimes is the only symptom observed. The use of thyroid extract relieves the symptoms, which indicates, I think, that the symptoms are not allergic. I am not one of those who insist that the basis of all chronic suppuration in the sinuses is allergy.

In regard to operation on the frontal sinus, Dr. Van Alyea appears to be more conservative than I am when he advocates the saving of bullar cells. I believe the easiest and best pathway to the frontal sinus by the intranasal route is through the bullar cells. I usually remove not only the bullar cells but the agger cells as well, and enlarge the nasofrontal opening laterally, anteriorly and posteriorly. The floor of the frontal sinus should be removed to the septum and laterally as far as possible without breaking into the orbital tissue. In my experience this usually results in a permanent intranasal opening. My preference for the intranasal operation on the frontal sinus, when feasible, lies not so much in preserving the mucosal lining—that is important—but in preventing the collapse of the soft tissues because of removal of their support which is done in the external procedure. Dr. Simpson advocates an external approach to the infected frontal sinus with removal of the frontal process of the superior maxillonasal bone and the orbital plate of the ethmoid bone, much as the late Dr. Lynch did. I believe this widespread removal of bone in the entrance to the frontonasal opening is poor technique.

In regard to Dr. Van Alyea's proposal to preserve the frontal duct, I have not seen many cases in which the nasofrontal duct was not involved in disease of the frontal sinus. In many cases I have found constricting reactive osteitis in this region. In many cases pyocele does not seem to be due to infection from previous mucocele but is produced by bony growth constricting the nasofrontal openings. I do not mean to imply that pyocele never is a secondary result of mucocele but it is, I believe, in many instances the direct result of infection and its effect on the bone.

Dr. Van Alyea did not obtain good results from intranasal instillation of penicillin. I agree when instillation is done by the Proetz method once a day by the doctor. Instillation of 2 cc. of penicillin into a sinus does not seem to accomplish more than lavage with physiologic sodium chloride solution. The sinusitis of an occasional patient will appear to be cleared up by the use of lavage with penicillin or even with saline solution. Results of instillation of penicillin can be improved if the patient does it himself several times a day by Hymes' modification of the Proetz technique. The poor results from penicillin therapy, in my experience, were due to the fact that in order to secure therapeutic effect the solution of penicillin must be maintained in contact with the mucosa for two or three hours. Apparently ciliary activity takes place even in the presence of serious disease so that solutions introduced but once daily

are removed before sufficient time has elapsed to produce a result. If the solution is introduced six or eight times a day by the patient himself, in certain instances a curative effect is produced. I do not say 100 per cent cure, but cure in as many as 10 per cent of cases is a relatively good result.

By hyperplastic ethmoiditis I mean nasal disease in which allergy and infection are mingled. While it is true that, as Dr. Van Alyea states, many polyps hanging in the nose do not originate in the ethmoid labyrinth, yet the cells in the ethmoid labyrinth are full of polypoid material. The postoperative application of radium, which tends to produce fibrosis and therefore increases the supporting structure in the tissue, is beneficial. Radium is not curative but it is worth while as it gives symptomatic relief and does tend to prevent recurrence after removal of polypoid tissue.

Dr. Shapiro asked whether unilateral ablation of the frontal sinus can be achieved. I believe it can be done if care is taken to eradicate the mucosa from, and bevel the edges of, the frontal sinus as originally suggested by Lillie. Proof that the sinus never reforms is difficult to get because patients do not wish to submit to an examination unless symptoms recur However, I have a patient who has been without symptoms for more than five years after this type of operation. It seems to me that the poor cosmetic result of ablation is not really an important consideration now that cosmetic procedures have improved. Furthermore, if a patient has been operated on several times with recurrence of infection after each procedure, he does not care how he looks if he can get rid of the infection, and for such a patient I think no other type of operation is indicated. After the infection has been absent for six months or a year, suitable reparative plastic work can be done. The coronal incision is valuable in an ablating procedure if the patient has not been operated on previously with resulting unsightly scarring. This incision gives more adequate exposure and allows better beveling of the superior and lateral bony margins of the sinus, so that no dead space remains. Ablation of the mucosa of the sinus and then allowing fibrosis to destroy the lumen of the sinus, I believe, cannot be depended on. I have seen cases in which secondary abscesses occurred in the center of the fibrous tissue with considerable discomfort and pain. I think there are many failures with this technique.

Dr. Salinger asked about indications for nasal antrostomy. I feel this operation should be carried out when infection in the antrum fails to clear up after one or two lavages, or when an individual after so-called cure with lavage has repeated recurrence of

antral disease. If the presence of an infected tooth root is excluded, patients get prompt objective and symptomatic relief by nasal antrostomy. It is a satisfactory operation.

Dr. Mundt stated that he secured good results in chronic maxillary sinusitis with the Caldwell technique without removal of the lining mucosa. I think this is true. The approach I employ is different but the end result is the same. If one is more skilled in the Caldwell technique and gets better results with it, I do not see why he should not use it. It is a little more extensive and requires longer hospitalization than some other procedures. Whether an intranasal window or a Caldwell approach is done, the end result depends on the adequacy of the window left behind.

Antihistaminic drugs merely give symptomatic relief; they are not a cure. As soon as administration is stopped the symptoms return. For that reason they are unsatisfactory as a rule because allergy is a lifelong affliction. Good initial results are obtained from administration of 50 mg. four times a day of almost any antihistaminic drug. As soon as the edema of the nasal mucosa subsides, the polyps recede unless secondary fibrosis of the polypoid tissue is present. After the maximal result is obtained I decrease the dose gradually in an attempt to find the smallest possible dose that will give symptomatic relief. One reason for the failure of "allergic" therapy is lack of recognition of physical allergy for which the precipitating stimuli are changes in temperature, emotional storms, etc. Antihistaminic drugs are helpful temporarily, but I think a more important use for them is in rapid diagnosis of an allergic or pathergic condition.

Dr. Fraser spoke of the use of lipiodol for diagnosis and penicillin for treatment of chronic sinusitis. At the Mayo Clinic lipiodol was tried by Dr. Pattee for diagnostic purposes in a large group of patients who gave a history suggestive of sinusitis. Roentgenograms were made at intervals. Twenty-four hours after instillation of lipiodol there was tremendous swelling of the lining membrane of the sinus. In addition, roentgenograms made after instillation of lipiodol did not appear to have much additional diagnostic value over roentgenograms made by the usual method.

The etiology of polyps originating in the antrum and coming into the nasal chamber through the natural ostium has not been solved. This type has a tendency to recur unless the mucosa at the site of origin is removed. Such a polyp occurs in a single antrum; the other paranasal sinuses do not show evidence of polyposis. A

large choanal polyp protruding into the nasopharynx has been mistaken for a nasopharyngeal fibroma. At the Mayo Clinic histologic study of these polyps and their mucosal attachments has not suggested a definite cause for their appearance even though search has been made for a neoplastic tendency in the sinus mucosa.

Meeting of Monday, January 5, 1948

THE PRESIDENT, DR. JOHN R. LINDSAY, IN THE CHAIR

Electrocardiogram Changes Following Laryngectomy*

(Abstract)

CARL A. JOHNSON, M.D.

PAUL HOLINGER, M.D.

AND

ALBERT ANDREWS, M.D.

This study was undertaken after one of us had noted the frequency of gallop rhythm following operation on the larynx for carcinoma. It was thought that there was more postoperative cardiac disturbance from this type of operation than from many other major operations. We were also interested in the study of pulmonary and cardiac reflexes, particularly as related to angina, in patients with permanent tracheotomy openings. None of the patients had a history of anginal attacks or had anginal attacks following surgery.

The limitations of the electrocardiogram as a means of study of cardiac function are recognized; changes may occur following many conditions and procedures in the absence of organic cardiac disease. In spite of this it was thought that some information might be obtained on immediate, late, reflex and toxic effects of this operation, as well as on the genesis of various components of the electrocardiogram.

All patients studied had had a previous biopsy diagnosis of carcinoma of the larynx, and were in the age group of 40 to 80 years. Some had evidence of cardiovascular disease prior to the

*From the Department of Clinical Research, St. Luke's Hospital, Chicago.

operation. The type of operations were laryngectomy and laryngofissure, depending upon the extent of the lesion and the age and condition of the patient. Anesthesia was given by intratracheal inhalation and consisted mainly of mixtures of ether, cyclopropane, and nitrous oxide. The duration of anesthesia varied from 150 to 285 minutes. Postoperative care did not differ from that following most major operations; penicillin was given routinely unless there were allergic contraindications, in which case sulfadiazine was substituted. Patients were fed a liquid diet through a retained stomach tube. Frequent aspirations of the trachea were made by suction. In those cases in which acute atelectasis developed from a bronchial plug, a hard blow over the area usually sufficed to relieve the condition.

In the group of 49 patients upon whom laryngectomy was performed there were no fatalities. The morbidity varied, the average hospital stay being from two weeks to one month. The electrocardiogram changes showed mainly changes in the amplitude and contour of the QRS complex, changes in the ST segment and changes in the T-wave. In some instances the changes were mainly in one lead, in others in more than one lead. The changes were not uniform, nor was the time of onset. In some the changes occurred shortly after operation; in others, several days later. In some cases changes were progressive until the patient left the hospital; in others the tracing had returned to the appearance of the control electrocardiogram. About 90 per cent of the patients showed these changes.

It is difficult to speculate on the causes of the various changes observed, particularly the late effects. The type and method of administration of anesthesia has been uniform, although the duration and the combination of anesthetic agents varied with each case. The early effects of ether in an ordinary operation are known to be premature beats, paroxysmal auricular tachycardia and disturbance of the pacemaker. However, no reference could be found on any late effects. Even less is known of the effects of the various combinations of anesthetic agents used in this series. So far as we are able to determine, the combinations used were chosen from clinical experience, not as a result of any scientific experiments on their late effects on the cardiovascular system. The procedure of administration of anesthesia is that of necessity; it does reduce the dead air space of the respiratory tract and little if anything is known as to whether this may be a factor in producing the changes observed. Other studies will be needed following other operations to determine if similar results are obtained. This study suggests the importance of a statistical study of the effects of individual anesthetic agents,

as well as various operative procedures, to determine what factors are responsible for the long recovery period.

One would surmise that the electrocardiogram changes observed in this series are the result of toxic, reflex and traumatic components, as well as an adjustment of the pulmonary physiology which follows this operation.

DISCUSSION

DR. PAUL HOLINGER: This subject has been of interest to us during the past three years since Dr. Johnson has assumed the medical management of our patients having laryngectomies. In this series of 49 laryngectomies, some of the operative procedures were relatively long. Some were simple laryngectomies using a clamp, others more radical operations with resection of the hyoid bone, the tracheal rings and the entire larynx, and there were also cases in which irradiation had been done previously but the tumor had recurred. The duration of the operation was not found to have any specific relationship to the degree of change noted in the electrocardiogram. The duration consisted of the total elapsed time from the start of anesthesia, not from the time of incision. Further, the type of anesthesia cannot be correlated to the changes. All anesthetics were given by the Department of Anesthesia through an intratracheal tube, using a tube in the trachea when the trachea is finally opened after dissection of the larynx. Further work must be done on other operative procedures and on various types of anesthesia in other operations involving the vagus nerve or its branches.

I think one of the important points of this paper is that we need and can have assistance in these cases. We allow the medical consultant to say when the patient is ready for operation and when he can get up and begin some activity after the operation. We rely on his findings, of which these studies that Dr. Johnson has presented form an important part, rather than on how the patient feels and what his temperature chart shows.

DR. ROBERT LEWY: I think it well worth while to emphasize the value of teamwork between the medical man and the surgeon. I am sure that in the long run morbidity and mortality rates will be influenced favorably. The electrocardiogram of a man I saw some years ago who had just recovered from a serious case of scarlet fever combined with streptococcic pneumonia showed changes essentially comparable to those shown here. It was more iso-electric than it had been prior to his illness, and the QRS showed less wide variations and the T-wave was considerably flattened in Leads II and III.

This suggests to me that any insult to the human organism, whether medical or surgical, would be responsible for causing these changes. We should study not only the variables involved from surgical procedures but also the influence of toxemia, infection and medical conditions, for a better perspective of the problem.

DR. ALFRED LEWY: In the days when intubations for laryngeal diphtheria were fairly common, a number of deaths occurred in the attempt at intubation, in some cases as soon as the epiglottis was palpated. It was attributed to cardiac reflex, but I do not believe anyone worked out the sequence. Perhaps one of the essayists can explain what happened.

DR. FRANCIS LEDERER: Laryngectomy imposes quite a trial on the patient. It is a formidable procedure in which there is not only a traumatic influence but a change in the entire physiologic picture, and it is not surprising to see these changes in the electrocardiogram. Dr. Lewy's remarks remind me that many diphtheritic patients died following tracheotomy as well as during intubation. I believe in such instances the patient had myocardial damage and death was due to this, not due to a reflex; that the patient had respiratory embarrassment and finally was subjected to this procedure.

DR. ALFRED LEWY: The type of case to which I referred was that in which death occurred almost immediately after the larynx was touched. I do not know whether in the presence of diphtheria there was myocardial damage, but the deaths were too sudden—it seemed as if there was a nerve reflex of some sort.

DR. JOHN R. LINDSAY: With the great development of chest surgery in the last couple of decades, one wonders whether in such procedures as removal of part of the esophagus and pneumonectomy there are similar changes in the electrocardiogram.

DR. CARL A. JOHNSON (closing): My first interest in this subject occurred while I was a medical student, when it came to my attention that some of the fraternities used a form of the Valsalva experiment in initiations. This consisted of having the subject take a deep breath and hold it while compression of the chest was applied. It was noted that unconsciousness could be produced easily by this procedure. Dr. Luckhardt and I did some work on dogs at the University of Chicago and confirmed other work to the effect that raised intrapulmonic pressure produces a marked fall in blood pressure and change in the heart rate, and in some dogs sudden death occurred. Sudden death could be prevented by anesthesia, morphine,

atropine, or section of the vagus nerves. This is in keeping with the work of Dr. Joseph Capps, who showed that sudden death during a thoracentesis could be prevented by one's giving morphine and atropine before the procedure. He also showed that the reflexes were exaggerated when the tissues were inflamed. The sudden death in infants, mentioned by Dr. Lewy, may have been on this basis.

Our study is only preliminary in nature and offers a method of study of operative and postoperative factors which have a bearing on morbidity. There has been no fatality in this series. One patient had delayed AV conduction and bundle branch block, and went through the operation easily, while others whose electrocardiograms looked more normal had difficulty. The significance is hard to evaluate, especially when one considers the electrocardiogram to be a composite of all electrical changes in the body at the instant of deflection. The major changes in the electrical record are, however, the result of cardiac action.

DR. A. H. ANDREWS (closing): In answer to Dr. Lindsay: in the work done by Ivy on section of the vagi, about 40 per cent of the dogs died immediately of asphyxiation. This could be prevented by administration of atropine and the heart could be started by contact massage. It has been thought for many years that sudden death due to stimulus about the pleura is based on this mechanism; in the presence of acute inflammation this particular reflex was more apt to occur.

The Vibration of Vocal Folds at Selected Frequencies*

(Abstract)

I. P. BRACKETT, PH.D.

The present study was concerned with the vocal fold vibration of two male subjects phonating at the frequencies of 256 c.p.s., 128 c.p.s., and a glide between 128 and 256 c.p.s. Vocal fold action at these frequencies was photographed at 4000 pictures per second. Results were obtained from a frame by frame analysis of the films. The analysis of the vocal fold action was achieved by studying the glottal openings during the cycle of vibration, the phase relationship of the cycle, the adjustment of the superior laryngeal structures, and the length of the glottis on successive frames of the cycle. All results

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were taken from composite tracings of the film image which was enlarged six times.

The results of the study indicate that the cycle of vibration consisted of three phases: closed, opening, and closing. After the closed phase of the cycle, the first observable opening appeared slightly anterior to the center of the folds and moved predominantly in an anterior to posterior direction as well as laterally during the opening phase of the cycle, and posterior to anterior during the closing phase of the cycle. At comparable frequencies and intensities, Subject I consistently had a larger glottal area than Subject II, but for both subjects the glottal area was greater for the frequency of 128 than for 256 c.p.s.

At the frequency of 256 c.p.s. both subjects had a phase relationship of one-third closed, one-third opening, and one-third closing. At 128 c.p.s. Subject I continued the same proportion of frames for the phases of the cycle, while Subject II had one-fourth of the frames for the closed phase, three-eighths of the frames for the opening phase and three-eighths for the closing phase. At the completion of the glide (frequency 256 c.p.s.) Subject I showed one-half of the frames for the closed phase and approximately one-fifth of the frames for the opening and closing phases of the cycle. There is evidence that differences in glottal area as a function of time and differences in the proportion of time for the three phases of the cycle may be related to the type of voice production.

For both subjects the area of the visible folds was greater at the frequency of 256 than at 128 c.p.s. During the glide, Subject I decreased the area of the observable folds between the frequencies of 128 and 256 c.p.s., while Subject II increased the area of the observable folds in phonating the same glide.

The glottal length for a given subject also appeared to vary at comparable frequencies and intensities. Differences in glottal length, glottal area, and the proportion of time for the phases of the cycle appeared to be related to the type of voice production.

The study reveals that further investigation is indicated to study the vibratory action of the folds during different types of voice usage.

DISCUSSION

DR. PAUL MOORE: You may wonder why a study of this kind should be made. It is one of several which have been completed and it is a part of a contemplated series dealing with the function of the

larynx during voice production. If we can discover how the normal and abnormal larynges produce sound, we may be able to develop more effective remedial programs.

There are some interesting implications in Dr. Brackett's study. One relates to the theories of vocal cord vibration. Although the present investigation involves a glottal opening, this space is directly related to the movement of the cords. The fact that they remain closed for a certain portion of each vibratory cycle and then open to emit a puff of air lends support to the "puff" theory. This theory receives further support from another finding in the study, namely, that the relative time occupied by the three phases of the vibratory cycle may vary without changing the pitch of the tone providing the glottis opens at the proper frequency.

Another point of interest related to pitch is the sizes of the larynges of the two subjects photographed. One subject was much larger than the other and had a larger larynx, yet both produced the same pitches. The photograph indicates that muscular tension differed considerably between pitches in the same subject and between the two subjects at the same pitches. There is a strong suggestion that laryngeal adjustments related to air pressure determine the rate of movement of the cords.

Some of the photographs also cause one to believe that loudness is directly related to the area of the glottis during the vibratory cycle, and to air pressure. Subsequent study may provide answers to such surmises.

In your practice you regularly see laryngeal pathology which produces defective voices. You also see patients, as we do, who have voice defects without demonstrable laryngeal deviations. We are vitally interested in both types and hope that our research will prove beneficial to our related fields. I should like to point out that the subjects used in Dr. Brackett's study were necessarily trained to tolerate and to manipulate a guttural mirror in a manner not possible for a naive subject. We are working toward more universal techniques and will welcome any suggestions you care to make.

DR. PAUL HOLINGER: I should like to compliment the essayists on this work. This is the sort of paper we are anxious to have presented before our group since it follows so logically the preliminary work presented last year. The extremely complicated and interesting technique of photography used leaves us very envious, and I should like to know if there are any tricks you have along that line about which you could tell us. We have had our greatest stumbling

blocks with mirror photography in these areas. The possibility of further study of the influence of deformity or the changes produced by various types of pathology comes to mind immediately. This method adds an entirely new approach to a problem which interests us all.

DR. WALTER H. THEOBALD: I wonder whether the cord changes from E to G string, for example, when he changes from 256 to 128 vibration.

DR. I. P. BRACKETT (closing): It is difficult to determine exactly the length of the vocal folds for different frequencies because the superior laryngeal structures often limit the view. It is commonly thought that the vocal folds increase in length as the pitch is raised, and on the films they appear to do so. The relationship between the length of the folds and the length of the glottal opening has not been determined. The ends of the glottis are not always visible during the opening and closing phases of the cycle, so that one cannot be certain that the ends of the glottis when the glottal opening is at its greatest, correspond to the ends of the vocal folds. There is evidence that the length of the glottis may not be related to the length of the folds at a given frequency. The type of voice production appears to affect the glottal area (length and width), although the exact relationship has not been determined.

There is substantial difference in tension of the vocal folds for different frequencies. At 128 c.p.s. you can see a definite "ripple", a sign of laxity, which starts anterior to the center of the folds and spreads across the superior surface of the folds until the glottis is at its widest. The "ripple" disappears as the folds begin the closing phase. At 256 c.p.s. the "ripple" cannot be seen. The vocal folds are definitely more taut at 256 than at 128.

DR. JOHN R. LINDSAY: What happens to the opening when you increase the intensity?

DR. BRACKETT: I cannot answer that. The speed and noise of the camera made it difficult to be certain of the exact intensity of the voice. The voice intensities varied slightly in the present research, but not enough to draw any conclusions. This research was an attempt to vary frequency at comparable intensities. I hope sometime to vary intensity at constant frequencies.

Abstracts of Current Articles

EAR

Speech Hearing In Otosclerosis.

Ersner, M. S., and Saltzman, M.: Arch. Otolaryng. 46:753-761 (Dec.) 1947.

The authors summarize their method of examining patients with a hearing loss and making the diagnosis. They emphasize that word-hearing tests give a different result from pure tone audiometry. The word-hearing loss is greater in otosclerosis than the audiogram would indicate. Likewise, they find that amplification is more effective in otosclerosis than in nerve deafness.

HILDING.

Nonvibratory Tinnitus.

Fowler, Edmund Prince: Arch. Otolaryng. 47:29-36 (Jan.) 1948.

The author describes a nonvibratory tinnitus and describes its mechanisms and the usual causes. He gives evidence for the reality of subaudible tinnitus. He outlines an approach to treatment consisting of twelve steps and encourages the otologist to spend more time and effort in connection with this annoying symptom.

He states that in the majority of instances the tinnitus can be brought below or to a little over the threshold of hearing, so that it no longer constitutes a major irritation.

HILDING.

NOSE

Nasal Hemorrhage.

Neivert, Harry, Engelberg, Recha, and Park, L. A.: Arch. Otolaryng. 47:37-45 (Jan.) 1948.

These authors studied 104 patients subject to epistaxis and found a high incidence among them of ascorbic acid and prothrombin deficiency. The concentration of ascorbic acid and prothrombin was normal in less than eleven per cent.

Gratifying clinical results were obtained with the administration of ascorbic acid and vitamin K. The authors conclude that vitamins C and K have a definite place in the management of nasal hemorrhage.

HILDING.

The Roentgen Treatment of Subacute Sinusitis In Children.

Laing, Donald R.: Radiology 50:52-56 (Jan.) 1948.

Children with an upper respiratory infection for more than five or six days should have nasal smears made to determine the presence of pus cells and eosinophiles. A single radiograph of the sinuses is of diagnostic value.

In the past ten years the author has given roentgen treatment for sinus infection to more than 4000 children between the ages of one and thirteen years. 900 were selected for this study, showing varying degrees of maxillary and ethmoid sinus infection of the subacute type.

Three treatments over a period of 8 days with a total of 240 r were given. X-ray factors for younger and older children are detailed.

71% of the 900 cases were cured after one roentgen treatment series.

15% showed moderate improvement. Repeating the treatment series in the remainder after 6 weeks' interval cured 55%. This gives complete recovery in 79% of the total group, a favorable result in 7% and temporary or no response in 14%.

Hufford reported that 56 children with hypertrophic lymphatic tissue causing eustachian tube block showed marked improvement or complete relief from deafness in 73 per cent of cases following roentgen therapy.

A. F. Rossitto reported comparable results in 600 cases of sinusitis.

Laing gives three treatments spaced over 8 days; the average dose is 80 r, making a total of 240 r as a series. Sulfonamides are not given during the treatment and he prefers not to give roentgen therapy to patients who have had a sulfonamide within the previous five to seven days.

The author has cures in approximately 80 per cent of the eustachian tube block cases.

JORSTAD.

Plastic Repair of the Obstructing Nasal Septum.

Fomon, Samuel, Gilbert, J. G., Silver, A. G., and Syracuse, V. R.: Arch. Otolaryng. 47:7-20 (Jan.) 1948.

The authors describe a new method of submucous resection to be done in connection with rhinoplastic surgery. It is better to do the rhinoplastic operation and the submucous operation separately three or more weeks apart. Under certain circumstances that may be impossible, and both must be done at the same sitting. When they can be done separately the submucous resection should be done first. Otherwise, there is sufficient spring in the septum so that the nasal pyramid after rhinoplastic surgery will not remain in place. When, for instance, there is a long nose with caudal displacement of the septum, and an "s"-shaped or deflected nasal pyramid, it is necessary to do both operations at one sitting.

HILDING.

Morphologic Deformities of the Lower Lateral Cartilages.

Daley, Jacob: Arch. Otolaryng. 47:49-63 (Jan.) 1948.

The author gives an excellent description of the anatomy of the lower lateral cartilage, together with its variations in size and shape. He describes the effects of these variations on the size and shape of the lobule. The surgical procedures for exposing the cartilage and trimming it to overcome the various types of deformity are given.

HILDING.

Nose and Skin Carriage of Staphylococcus Aureus in Patients Receiving Penicillin.

Moss, Brenda, Squire, J. R., and Topley, Elizabeth: Lancet, Feb. 28, 1948.

The authors assert that many people persistently carry staphylococcus aureus in the nose for many weeks. It is also found on normal skin particularly on the back of the wrist in a smaller but still high proportion of persons. More "skin positive" persons show nasal carriage than do "skin negatives." This leads to the belief that staphylococci found on the normal skin are usually derived from the nose, probably by direct contact.

The authors applied penicillin locally in the nose by spray and a nasal cream in a selected group of hospital patients with normal skins who were persistently carrying coagulase-positive staphylococci

in the nose. They believe that most of the staphylococci in the nose are carried in the skin-lined vestibule and that the mucosa-lined cavities of the nasal fossae are relatively sterile.

Each patient was given a ten-day course of 50,000 to 100,000 units of penicillin in a spray applied thrice daily from an atomizer whose removable sterile glass end was placed just inside the nostril. When a cream was used on eleven patients, it was applied twice daily with swabs in a concentration of 100,000 units per gram in a Lanette wax base. The results of this treatment were that in a group of 21 patients the nasal carriage rate fell from 97 per cent before treatment to 67 per cent in the first five days and to 37 per cent in the next five days of intranasal penicillin therapy. In contrast 20 untreated patients had a nasal carriage rate which did not vary during the hospital stay and was not significantly different from the intranasal penicillin series before treatment was started.

On the other hand systemic penicillin in a daily dosage of 100,000 units failed in 15 surgical patients to influence the nasal or skin carriage of staphylococcus aureus. This failure is explained on the hypothesis that the site of the staphylococcus aureus on the epithelium of the skin or nasal vestibule is not reached by any transudate from the capillary blood.

Their experimental finding that the reduction of nasal staphylococcus aureus carriage is associated with a significant fall in carriage on the wrist skin strongly suggests that the nose was the predominant source of the staphylococcus aureus on the wrist.

GROVE.

PHARYNX

Histologic Changes In Squamous-Cell Carcinoma of the Mouth and Oropharynx Produced by Fractionated External Roentgen Irradiation.

Hall, John W., and Friedman, Milton: Radiology 50:318-350 (March) 1948.

A study of material derived from 28 patients with squamous-cell carcinoma of the mouth and oropharynx is the subject of this contribution. During the irradiation periodic biopsies were made with the following results:

"1. The destructive effect of radiation on squamous-cell carcinoma of the mouth and pharynx is achieved by several processes: acute cell death, progressive enlargement of cells to giant-sized tumor cells, and radiation keratogenesis.

"2. Acute cell death is the most common change. It affects the cytoplasm, nucleus, and other components of a cell impartially and is usually seen in the first seven days. This is the process whereby the most radiosensitive cells are destroyed. Its importance has not been sufficiently emphasized.

"3. The less sensitive cells, not undergoing acute cell death, show progressive enlargement to giant-sized tumor cells. This is accomplished through two different mechanisms: (1) swelling and vacuolation of the cytoplasm and to a lesser extent of the nucleus, probably through a physical-chemical disturbance of the intercellular and intracellular exchange of fluids; (2) disruption of the nuclear function, resulting in the formation of various nuclear abnormalities.

"4. Radiation keratogenesis, another mechanism of cell destruction, is the acceleration either in time or degree of the normal process of keratinization. It is a cytoplasmic phenomenon.

"5. Under-irradiation of a tumor (chiefly in the form of an inefficiently small daily dose) produces some destruction of tumor cells, but chiefly provokes a maturation of the more radioresistant cells.

"6. The most pronounced change in the stroma is replacement fibrosis, secondary to and in proportion to radiation destruction of a tumor. Irradiation also increases hyalinization of the connective-tissue stroma, and, to a slight extent, produces degenerative changes within some fibroblasts, namely swelling and vacuolation.

"7. With fractionated irradiation, damage to the blood vessels is usually slight, occasionally moderate in degree. From the material studied, no conclusion can be drawn as to the relationship between radio sensitivity of the tumor and vascular damage.

"8. A single biopsy, taken between the seventh and eleventh day during a course of fractionated irradiation, will yield considerable information concerning the radiosensitivity of the tumor and the effectiveness of the irradiation technic."

[Authors' summary]

Tonsillectomy And Adenoidectomy. Unsatisfactory Results Due to Chronic Maxillary Sinusitis.

Walker, F. M.: Brit. Medical Journal, Dec. 6, 1947, pp. 908-910.

Walker reports on 1779 tonsillectomies and adenoidectomies performed in Ayrshire during the year 1946. Of these patients 442

or 25 per cent were considered to have sufficient signs or symptoms to warrant antrum puncture. Of these 442, 106 showed no pus in the washings. In the remaining 336 patients the washings were grossly purulent, mucopurulent or mucoid. Thus in 19 per cent of the entire 1779 cases there was evidence of chronic maxillary sinusitis, although only in 290 cases or 16 per cent could actual purulent secretion be washed out of the antrum.

Cervical adenopathy was twice as common in uncomplicated cases of enlarged tonsils and adenoids, while spasmodic cough and night terrors were five times more common in the latter group.

Of the 290 patients in whose antra purulent secretions were encountered, 29 failed to report, 133 were completely cured and 128 showed unsatisfactory results. Before 1946 the Proetz displacement treatment with 0.5% ephedrine in physiologic sodium chloride solution gave some satisfactory results but this was found to be an unsatisfactory form of treatment for the great majority of the patients because of the large area over which they were scattered and the need of repeated treatments. Instillation of sulfex and penicillin (10,000 units in 1 ml. of fluid) into the antra after irrigation was not found to be more efficacious than simple irrigation alone. If the antrum infection did not clear up in six irrigations the children were admitted to the hospital for a double antrostomy.

Walker's conclusions were that "an analysis of the 1779 cases operated upon for tonsils and adenoids has shown that unsatisfactory results are due, in the great majority of instances, to the presence of chronic sinusitis, which condition in children is often associated with night terrors, restless sleep, chronic cough, headache and pyrexial attacks of short duration.

GROVE.

Experiences with Nasopharyngeal Carcinoma.

Graham, Vincent P., and Meyer, Ralph R.: Radiology 50:83-91 (Jan.) 1948.

The authors report on 29 patients with carcinoma of the nasopharynx seen between 1932 and 1942. Age limits were 13 to 72 years. Over one half of these patients were under the care of a physician and had one or more nasal operations directed toward the relief of symptoms without recognition of the primary lesion.

In 62 per cent, the chief complaint on admission was a cervical mass. Twenty-six patients received x-ray therapy. Eighteen of

the 26 patients had palpable cervical lymph nodes and in four of these the node enlargement was bilateral.

Eighteen of the 24 patients examined by axial roentgenograms of the skull showed bone destruction, most frequently in the region of the petrous apex and the foramen ovale. Transitional cell carcinoma was the commonest lesion.

On the average, a patient received a depth dose of 5000 r x-radiation to the nasopharynx in 40 days, through portals. Six case reports and eight figures to show radiographic changes in the base of the skull are included.

Although these patients came for treatment in an advanced stage, the authors conclude that good palliation and prolongation of life were obtained in almost all cases and in about 20 per cent a cure was effected by radiotherapy.

JORSTAD.

Retropharyngeal Lipoma.

McFarland, J. C.: Lancet, Feb. 28, 1948.

McFarland reports a case of a three-month-old child admitted to the hospital with a six weeks' history of difficult breathing and occasional apnea. Food was taken with great difficulty. A lump on the left side of the pharynx suggested a retropharyngeal abscess. Incision did not reveal pus. The mass felt hard to the touch. Four days later it was excised and microscopic examination revealed an encapsulated lipoma. Lipoma of the pharynx is relatively rare, only 35 previous cases having been reported by Figi and Hunt.

GROVE.

Glossopharyngeal Zoster, Followed by Varicella in Two Contacts.

Wright, Frederick J.: Lancet, Dec. 27, 1947, pp. 946-947.

Wright reports a case of zoster in the distribution of the glossopharyngeal nerve. He states that the ganglia connected with the 5th, 7th, 8th, 9th and 10th cranial nerves are developed from the neural crest and are thus homologous with the spinal posterior root ganglia. It, therefore, seems probable that the superior and petrous ganglia of the 9th cranial nerve are primarily involved in glosso-

pharyngeal zoster and in his case the jugular ganglion and the ganglion nodosum may also have been involved.

The eruption in glossopharyngeal zoster is seen as an ipsilateral involvement of the pharynx, tonsillar fossa, faucial pillars and soft palate.

Wright's case is of a male, 47 years of age, whose condition began with pain in the throat and right ear for five days. When seen on the sixth day his temperature was 100° F. There were several ovoid ulcers on the posterior pharyngeal wall to the right of the midline. On the following day a cluster of superficial ulcers of various shapes appeared on the right side of the soft palate. These were arranged in groups. On the following day other spots were noted, one on the nose in the midline, most of them on the right side of the chest and right arm, but a few to the left of the midline. In about two weeks he was practically normal again as to the eruption, although nausea and vomiting continued for ten days more.

About 17 days after the onset of this man's illness, his daughter, aged 13, and two days later her brother, aged 4 years, exhibited typical varicella rashes. Wright's belief is that the glossopharyngeal zoster with probable involvement, also, of the right vagus nerve was due to the virus of varicella with which his patient's children became infected as contacts.

GROVE.

LARYNX

Laryngocele.

McLaurin, J. W.: Laryngoscope 57:616-622 (Sept.) 1947.

McLaurin reports a case of laryngocele in a man whose only symptom was a mass the size of a walnut on the left side of the neck. The growth increased in size with swallowing. The patient was given intratracheal cyclopropane oxygen anesthesia and an incision was made one inch below the border of the left mandible, extending from the angle almost to the midline. The laryngocele came into view as the platysma and deep fascia were incised; it was located anterior to the sternocleidomastoid muscle and just beneath the submaxillary gland.

The sac was removed from its attachment to the thyrohyoid membrane in the midline, above the thyroid cartilage, and the stump tied with heavy silk.

The cyst contained air and 4 cc. of yellowish fluid, and the lining showed inflammatory changes of a chronic nature. Healing was uneventful and complete.

VAN ALYEA.

Congenital Laryngeal Web: Its Eradication.

Poe, D. L., and Seager, P. S.: Arch. Otolaryng. 47:46-48 (Jan.) 1948.

The authors describe a method of dealing surgically with a congenital laryngeal web. It consists essentially in the introduction of a polished light metal plate between the edges of the divided web. The plate is secured in position by a silver wire which penetrates the larynx anteriorly in the midline. It is left in place for a period of ten days, after which, in a case which they report, there was no evidence of any recurrence of the web.

HILDING.

BRONCHI

The Visible Bronchial Tree.

Fleischner, Felix G.: Radiology 50:184-189 (Feb.) 1948.

Three figures demonstrate differences in radiopacity between air-filled and paraffin-filled paper drinking straws. Eight figures show differences in radiopacity of lung markings interpreted in the light of this experimental finding.

Visibility of the air-filled bronchi within parenchymatous lesions of the lung is emphasized as a diagnostic sign. Absence of this visible "bronchial tree" does not rule out pneumonic consolidation. Failure to demonstrate the bronchi may be due to filling with secretions, edema, or blood, or it may be due to over- or underexposure of the film.

This sign has been found most valuable in pediatric roentgenology.

JORSTAD.

ESOPHAGUS

Dissecting (Intramural) Pharyngo-Esophageal Diverticulum.

Melamed, Abraham, and Walker, Lynn J.: Radiology 49:712-715 (Dec.) 1947.

This is a case report of a 15-year-old boy with history of difficulty in swallowing solid food for one month. Three roentgen views following "barium swallow" are shown. Gradual disappearance of the barium in a diverticulum at level of the sixth and seventh cervical vertebrae following "blank swallowing" is emphasized. Its completely intramural location in the midline posteriorly was determined at the time of operation. The sac had not increased appreciably in size between the time of examination and the operative procedure nine months later. Very close scrutiny of the lateral projection reveals a pyriform shadow of relatively greater density than the barium-filled esophagus. This is suggestive of the intramural character. Postoperative roentgen examination of the pharyngo-esophageal area revealed no abnormality at time of hospital discharge five and one-half weeks following admission.

JORSTAD.

MISCELLANEOUS

Histamine in Certain Types of Headache.

Salinger, Samuel: Wisconsin M. J. 47:382-385 (April) 1948.

In this paper Salinger deals with two types of headache, that which is due to involvement of sensory nerves or ganglia and that due to vascular tension. Differentiating the strictly neurogenic and the ganglion cell headaches from those of vascular origin should be a simple matter if we remember that the former are experienced in the area of distribution of certain definite cranial nerves and ganglia, whereas the latter follow the distribution of the arteries.

Distention of the endocranial blood vessels or traction and distortion will produce a headache which radiates along the distribution of the vessel. Aside from expanding lesions of the brain and its appendages we find that the causes of vascular headache may be of allergic, endocrine, hypertensive or neuropsychogenic origin.

Histamine cephalalgia usually occurs in middle life. The attacks come on at night and are of short duration but oft repeated. The pain is severe, focuses in and around the orbit, radiating throughout one side of the head, face and neck and is accompanied by nasal stuffiness, watery discharge and lacrimation. This syndrome has

been shown to be due to histamine and can be induced by the hypodermic injection of histamine.

Migraine usually comes on early in life, has certain prodromal symptoms which are followed by severe hemicrania accompanied by nausea and vomiting. These symptoms are all due to sudden changes in the vascular tension within the cranium caused by a disturbance of the autonomic nervous system.

Headaches due to nervous tension can usually be traced to some emotional upset and persist only as long as the disturbing factor is present.

Psychogenic headaches, on the other hand, are closely linked to deeper personality changes, are more diffuse in character and vague as to distribution and occurrence. In most cases the patient's appearance belies the severity of the pain complained of.

In patients with physical allergy the headaches and other allergic phenomena are produced by changes in temperature, extreme heat and cold, drafts, chilling and the like. The pain is often referred to muscles about the head and can be termed a myalgia. It is often accompanied by local tenderness over the insertion of muscles about the head and at the base of the skull.

In all of the types of headache outlined above sudden changes in vascular tension due to irritation of the autonomic nervous system constitute the fundamental constitutional factor and since in many cases they are similar to typical specific allergic reactions, it is generally believed that histamine plays a vital role.

The allergic character of most of these headaches is shown by the fact that relief frequently follows the administration of histamine.

Salinger favors the minimal subcutaneous method of administration with a daily or twice a day increase in the dose to the point of tolerance over the more massive intravenous method proposed by Horton. The tolerance dose is continued at four to seven-day intervals for a few weeks until symptoms disappear.

In regard to the skin sensitivity histamine test, Salinger feels that it is not entirely reliable because a negative reaction, in which the patient does not exhibit a large wheal with pseudopods, swelling and itching, does not mean that the patient is histamine-negative. Salinger cites case histories of the various types of headache discussed and the reaction to histamine therapy.

GROVE.

A Subsequent Report On Roentgen Therapy in Carotid Sinus Syndrome.

Stevenson, C. A., and Moreton, R. D.: Radiology 50:207-210 (Feb.) 1948.

This is a report 24 cases, including five from the 1939 report, of carotid sinus syndrome in which roentgen therapy was used as the sole therapeutic agent. All the patients showed duplication of their attack when pressure was applied over one or both carotid sinuses.

X-ray factors used are 200 kv constant potential, 1 mm. copper and 1 mm. aluminum filter, 50 cm. target skin distance; 400 r measured in air, is given in one dose to one side of neck; the opposite side is given the same treatment on the following day. A 10x10 cm. field is usually adequate.

The age of the patients was 27 to 71 years; two tables are included which give essential data on each patient. Most patients received two or more treatments.

Forty-four per cent obtained complete relief and 25 per cent partial relief. Observation extended from one month to eight years.

JORSTAD.

Chemical Meningitis Following the Use of Tyrothricin.

Otenasek, F. J., and Fairman, David: Arch. Otolaryng. 47:21-28 (Jan.) 1948.

The authors report two cases in which a severe chemical meningitis developed suddenly after a nasal sinus had been irrigated with a suspension of tyrothricin. Both patients developed violent acute meningeal symptoms from which both recovered. Later both developed internal hydrocephalus due to the chronic thickening of the meninges with blockage of the ventricular system. One patient died from this. Experiments were done on animals demonstrating that the pathologic process consisted essentially of chemical meningitis followed by thickening and fibrosis of the meninges.

HILDING.

Eosinopenia Caused by Adrenaline Infusion and by Insulin Hypoglycaemia.

Godlowski, Z. Z.: Brit. Med. Journal, Jan. 10, 1948, pp. 46-48.

In a previous communication the author found that insulin hypoglycaemia had a beneficial effect on all but one of a series of cases of allergic bronchial asthma. These cases were characterized by a fall in the eosinophil count of the blood. The return of the blood eosinophils to normal took place in an oscillating fashion.

During the present investigation Godlowski studied 11 allergic and eosinophilic subjects (9 bronchial asthma, 1 urticaria and 1 dermatitis).

An intravenous adrenalin infusion was used (8 to 10 mg. of adrenalin in 200 to 300 cc. of physiologic sodium chloride solution at the rate of 6 to 12 drops of solution per minute over a period of 8 to 12 hours). The eosinophils were counted each 30 minutes for the first 3 hours.

Two or three days later the insulin test was made, after a 12-hour fast, by injecting 100 units of soluble insulin subcutaneously and the same eosinophil estimations were made as with the adrenalin infusion.

The first six patients with bronchial asthma and the one with dermatitis were subjected to sternal puncture before and during the eighth hour of adrenalin drip and insulin hypoglycaemia, and the bone marrow studied for eosinophils.

In 1910 Bertelli and others produced a considerable eosinopenia in dogs by the hypodermic or intravenous injections of adrenalin. Cannon and others, experimentally, and Heilbrun and Libert, clinically, found that insulin hypoglycaemia is characterized by the liberation of adrenalin in an amount sufficient to meet the emergency of the rapidly falling blood sugar. These observations suggest that the eosinopenia occurring during insulin hypoglycaemia is due to an outpouring of adrenalin.

Godlowski summarizes his experiments as follows:

1. The peripheral blood eosinophilia decreases markedly and to the same extent during prolonged intravenous adrenalin infusion as during insulin hypoglycaemia.

2. The quantitative and qualitative similarity of this phenomenon suggests a common cause for the eosinopenia.

3. Adrenalin does not affect the eosinophilic content of bone marrow and the latter, accordingly, is not the site of eosinophilic retention.

4. The ability of the organism to secrete adrenalin to meet the emergency of hypoglycaemia might explain the improvement affected in allergic conditions by treatment with insulin hypoglycaemia.

GROVE.

Streptomycin and Tuberculous Meningitis in Children: Preliminary Note.

Debré, Robert, St. Thierry, Brissand, Ed., and Noufflard, H.: Brit. Med. Journal (Dec.) 1947.

This work was done at the Hôpital des Enfants Malades in Paris by a team of pediatricians, ophthalmologists, neurosurgeons, pathologists, bacteriologists and chemists. The authors stress the importance of early diagnosis in children. In children tuberculous meningitis often becomes apparent at an early stage of the infection. Latent tuberculous meningitis is not uncommon in acute or subacute miliary tuberculosis; therefore, in these cases the authors stress the importance of lumbar puncture as a routine procedure, at least in all cases in which there is the slightest doubt. The examination of the fundus oculi for choroidal tubercles or early disc changes is also important. The decision to employ streptomycin therapy should not be dependent on the presence of the tubercle bacillus in the spinal fluid alone for of 90 definite cases of tuberculous meningitis these bacilli were found in only 41 cases. Nor should the decision be based upon a negative Pirquet reaction for in 4 of the 90 cases this reaction was negative.

In the decision to employ streptomycin therapy one should consider the following factors: history, knowledge of recent contact, mode of onset, evolution, cytochemical abnormalities in the cerebrospinal fluid and even, when necessary, the ventricular fluid. They found very valuable cultures of the cerebrospinal fluid on Loewenstein's medium.

The authors believe that it is useless to start streptomycin therapy in cases which seem hopeless and they list coma as one sign which is invaluable to determine the hopelessness of the case. In their early treatment they used massive doses of 100,000 to 200,000 units per kilogram of body weight per day by both the intraspinal and the systemic route. With experience they found that the use of these massive doses over a prolonged period was dangerous in that it induced severe toxic disturbances. These consisted of general toxic disturbances, cutaneous reactions and meningeal reactions.

Their present mode of treatment consists of two phases: The "attack" phase consisting of large doses (100,000 units per kilogram per day, given parenterally, and at the same time two intraspinal injections per day of 50,000 to 100,000 units). This treatment is continued for one week; the "maintenance" phase in which the treatment is continued at doses below 50,000 units per kilogram per day, given parenterally, and no intraspinal injections.

The authors regard a weekly examination of the cerebrospinal fluid with special reference to the gradual fall of the lymphocyte count as an important indication of the efficacy of the treatment.

At the time of the writing of this paper 46 chidren out of a total of 70 with tubercular meningitis, who have undergone treatment for from two to ten months, are behaving like normal beings.

GROVE.

Notices

FOURTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

The British Association of Otolaryngologists is organizing the Fourth International Congress of Otolaryngology, to be held in London from July 17 to July 23, 1949. There will be further meetings, for those who wish to go, at Oxford, Cambridge and Edinburgh on July 25 and 26. It is hoped that a full academic program will be arranged, and also various social functions.

The Secretaries of the national otolaryngological societies have been circularized and asked to send a list of their members for individual notification. Should any Association not receive this letter, they should communicate with the General Secretary, Mr. F. C. W. Capps, F.R.C.S., 45, Lincoln's Inn Fields, London, W.C. 2.

ACADEMY HOME STUDY COURSES

The Home Study Courses sponsored by the American Academy of Ophthalmology and Otolaryngology, in the basic sciences related to these two specialties, will be given again, beginning September 1, 1948. Registrations must be completed before August 15. Detailed information may be secured from Dr. William L. Benedict, Executive Secretary, 100 First Avenue Building, Rochester, Minnesota.

STATE UNIVERSITY OF IOWA

The Departments of Speech, Psychology and Otolaryngology of the State University of Iowa will offer two special courses during the 1948 summer session. The first is a four weeks' intensive course in audiometry and the fitting of hearing aids and is open to anyone

who can meet college entrance requirements. The course will run from June 21 to July 17. The second course is an advanced seminar open to graduate students who have completed a first course in audiometry or who have had experience in public school or clinical testing. This course runs for eight weeks beginning June 9.

In addition to the two special courses a series of four lectures and round-table discussions will also be presented by authorities in the field of speech pathology and hearing conservation. Inquiries may be addressed to The Speech Clinic, State University of Iowa, Iowa City, Iowa.

BIOLOGICAL PHOTOGRAPHIC ASSOCIATION

The Biological Photographic Association will hold its convention at Houston Hall, University of Pennsylvania, Philadelphia, September 8-10, 1948. There will be an exhibition of prints, color transparencies and motion pictures in the field of biological photography; organized symposia and demonstrations are planned. For further information address The Secretary, Biological Photographic Association, University Office, Magee Hospital, Pittsburgh 13, Pa.